NATIONAL CITIZENS INQUIRY

COMMISSION D’ENQUÊTE NATIONALE CITOYENNE

BOOK OF EXHIBITS OF EXPERT WITNESS DENIS RANCOURT, PHD

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Denis Rancourt, PhD
https://denisrancourt.ca/

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Brief Report |
9 February 2023

Age-stratified COVID-19 vaccine-dose fatality rate for Israel and Australia

Denis G. Rancourt,[1,*] PhD ; Marine Baudin,[2] PhD ; Joseph Hickey,[1] PhD ; Jérémie Mercier,[2] PhD

[1] Correlation Research in the Public Interest (correlation-canada.org)
[2] Santé Liberté OÜ (jeremie-mercier.com)
* denis.rancourt@gmail.com

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https://ocla.ca/
https://www.jeremie-mercier.com/

ABSTRACT: It is now well established from autopsy studies and adverse effect monitoring that the COVID-19 vaccines can cause death. The vaccine-dose fatality rate (vDFR), which is the ratio of vaccine-induced deaths to vaccine doses delivered in a population, has recently been measured by us to be as large as 1 % in India and when "vaccine equity" campaigns were applied in high-poverty states of the USA, and to be 0.05 % in Australia, with data that is not discriminated by age group. Here, we provide the first empirical evaluations of age-stratified vDFRs, using national all-cause mortality and vaccine rollout data, for Israel and Australia. We find that the vDFR increases dramatically with age for older adults, being exponential with a doubling time of approximately 5.2 ± 0.4 years. As a result the vDFR is an order of magnitude greater in the most elderly population than the all-population value, reaching 0.6 % for the 80+ years age group in Israel and 1 % for the 85+ years age group in Australia, compared to < 0.01 % for young adults (< 45 year olds). Our results imply that it was reckless to prioritise vaccinating those deemed to be in greatest need of protection.

It is well established that the COVID-19 vaccines can cause death, as seen from:

- detailed autopsy studies (Choi et al., 2021; Schneider et al., 2021; Sessa et al., 2021; Gill et al., 2022; Mörz, 2022; Schwab et al., 2022; Suzuki et al., 2022; Tan et al., 2022; Yoshimura et al., 2022; Onishi et al., 2023),
- adverse effect monitoring (Hickey and Rancourt, 2022),
- a recent survey study (Skidmore, 2023),
- studies of vaccine-induced pathologies (e.g., Goldman et al., 2021; Kuvandik et al., 2021; Turni and Lefringhausen, 2022; Edmonds et al., 2023; Wong et al., 2023), and
- more than 1,250 peer-reviewed publications about COVID-19 vaccine adverse effects (React 19, 2022).

In particular, a study of the Vaccine Adverse Event Reporting System (VAERS) data for the USA showed that the COVID-19 injections can be understood as individual challenges to the body, and that "toxicity by dose" is a good first-order model of the phenomenon for the adverse effect of death (Hickey and Rancourt, 2022). An exponential increase of lethality with median age of those dying following injection was observed (Hickey and Rancourt, 2022).

There is also the known vaccine injury compensation programmes of states worldwide, which include death resulting from the COVID-19 vaccines (Mungwira et al. 2020; Wood et al., 2020; Crum et al., 2021; Kamin-Friedman and Davidovitch, 2021). Japan, Canada and the UK have granted compensation for COVID-19 vaccine induced deaths (*The Japan Times*, 26 July 2022; Corbett, 6 September 2022; Wise, 2022).

We are pursuing a research program to quantify the vaccine-dose fatality rate (vDFR), which is the ratio of vaccine-induced deaths to vaccine doses delivered in a population. We do this at the population level of states, using epidemiological methods applied to all-cause mortality (ACM) and vaccine rollout data, by time (day, week, month), by jurisdiction and by age group (Rancourt et al., 2022a; Rancourt et al., 2022b; Rancourt, 2022).

Here we report our first age-stratification results.

We recently demonstrated that the COVID-19 vaccine rollouts caused significant increases in mortality in India, the USA, Australia, and Canada (see Rancourt et al., 2022a; and references therein).

Rancourt showed that the vaccine rollout in India (350 million doses) synchronously caused 3.7 million excess deaths, corresponding to a vDFR of 1 %; and provided

comprehensive reasons for concluding a causal relation to the vaccine rollout rather than coincidence involving other causes (Rancourt, 2022).

Our work on the Australian data established a non-age-stratified (all-population) mean vDFR of 0.05 %, in a phenomenon of step-wise increase in mortality synchronous with the vaccine rollout, which was also present in each of the eight states of Australia and in each of the age groups of the most elderly residents (Rancourt et al., 2022a).

Such determinations of vDFR are possible — despite the inherent difficulty in assigning cause to excess mortality, especially despite the difficulty in discerning excess mortality caused by the imposed pandemic-response conditions (or "COVID-19 conditions") — in two kinds of circumstances:

i. Jurisdictions in which there is essentially no measurable excess integrated ACM in the pre-vaccination period of the declared pandemic (typically 11 March 2020 to 1 January 2021),[1] followed by a large and sudden step-wise increase in ACM by time, synchronous with the vaccine rollout in the jurisdiction, and sustained through multiple-dose cycles of vaccination (e.g., Australia, India, Israel).
ii. Cases in which a specific vaccine rollout (e.g., first booster in Australia, "vaccine equity" campaign in the USA, first-dose in Ontario) is synchronous with an anomalous peak in ACM, which is not confounded by occurring at a seasonal peak position inferred from the historic trend.

In all these cases, which we have studied, the vaccine rollouts occur at significantly different times, for different jurisdictions and age groups, yet are always synchronous with the step-wise increases and anomalous peaks in ACM. In this regard, the graphs in our most recent paper and its appendices are compelling (Rancourt et al., 2022a; their figures 1A through 1D, 2, 4, 6A through 6D, 7, 8 and 9; their appendix figures A1-F1 (9 panels) and A2-F1), as are the graphs for India (Rancourt, 2022).

[1] The World Health Organization (WHO) declared a pandemic on 11 March 2020 (the "declared pandemic"). Vaccine rollouts typically did not start until late December 2020 and early January 2021, although several national jurisdictions had significantly later starts.

In addition, the all-population vDFRs, for individual states and for individual anomalous peaks in ACM, are all comparable in magnitude, in the range of approximately 0.03 % − 1 % (Rancourt et al., 2022a ; Rancourt et al., 2022b ; Rancourt, 2022).

The robust criteria described by Ioannidis (2016) for proving causality are amply satisfied:

- **Experiment:** The same phenomenon is independently observed in distinct jurisdictions, for distinct age groups, and at different times, which constitutes ample verification in independent real-world large-scale experiments.
- **Temporality:** The many step-wise increases and anomalous peaks in ACM are synchronous with vaccine rollouts, and the peaks in ACM have the same shapes and widths as the synchronous peaks in vaccine dose delivery by time; including in jurisdictions in which excess integrated mortality did not occur until vaccination was implemented after approximately one year of the declared pandemic.
- **Consistency:** The phenomenon is qualitatively the same and of comparable magnitude in each occasion in which it is observed.

Here, we perform the age-stratification analysis for Australia, and we add Israel.

Our method for quantification of vDFR by age group (or all-population) is as follows (Rancourt et al., 2022a):

i. Plot the ACM by time (day, week, month) for the age group (or all-population) over a large time scale, including the years prior to the declared pandemic.
ii. Identify the date (day, week, month) of the start of the vaccine rollout (first dose rollout) for the age group (or all-population).
iii. Note, for consistency, that the ACM undergoes a step-wise increase to larger values at the date of the start of the vaccine rollout.

iv. Integrate (add) ACM from the start of the vaccine rollout to the end of available data or end of vaccinations (all doses), whichever comes first. This is the basic integration time window used in the calculation, start to end dates.

v. Apply this window and this integration over successive and non-overlapping equal-duration periods, moving as far back as the data permits.

vi. Plot the resulting integration values versus time, and note, for consistency, that the value has an upward jog, well discerned from the historic trend or values, for the vaccination period.

vii. Extrapolate the historic trend of integrated values into the vaccination period. The difference between the measured and extrapolated (historic trend predicted) integrated values of ACM in the vaccination period is the excess mortality associated with the vaccination period.

viii. The extrapolation, in practice, is achieved by fitting a straight line to chosen pre-vaccination-period integration points.

ix. If too few points are available for the extrapolation, giving too large an uncertainty in the fitted slope, then impose a slope of zero, which amounts to using an average of recent values. In some cases, even a single point (usually the point for the immediately preceding integration window) can be used.

x. The error in the extrapolated value is overwhelmingly the dominant source of error in the calculated excess mortality. Estimate the “accuracy error” in the extrapolated value as the mean deviation of the absolute value difference with the fitted line (mean of the absolute values of the residuals) for the chosen points of the fit. This error is a measure of the integration-period variations from all causes over a near region having an assumed linear trend.

xi. Apply the same integration window (start to end dates during vaccination) to count all vaccine doses administered in that time.

xii. Define vDFR = (vaccination-period excess mortality) / (vaccine doses administered in the same vaccination period). Calculate the uncertainty in vDFR using the estimated error in vaccination-period excess mortality.

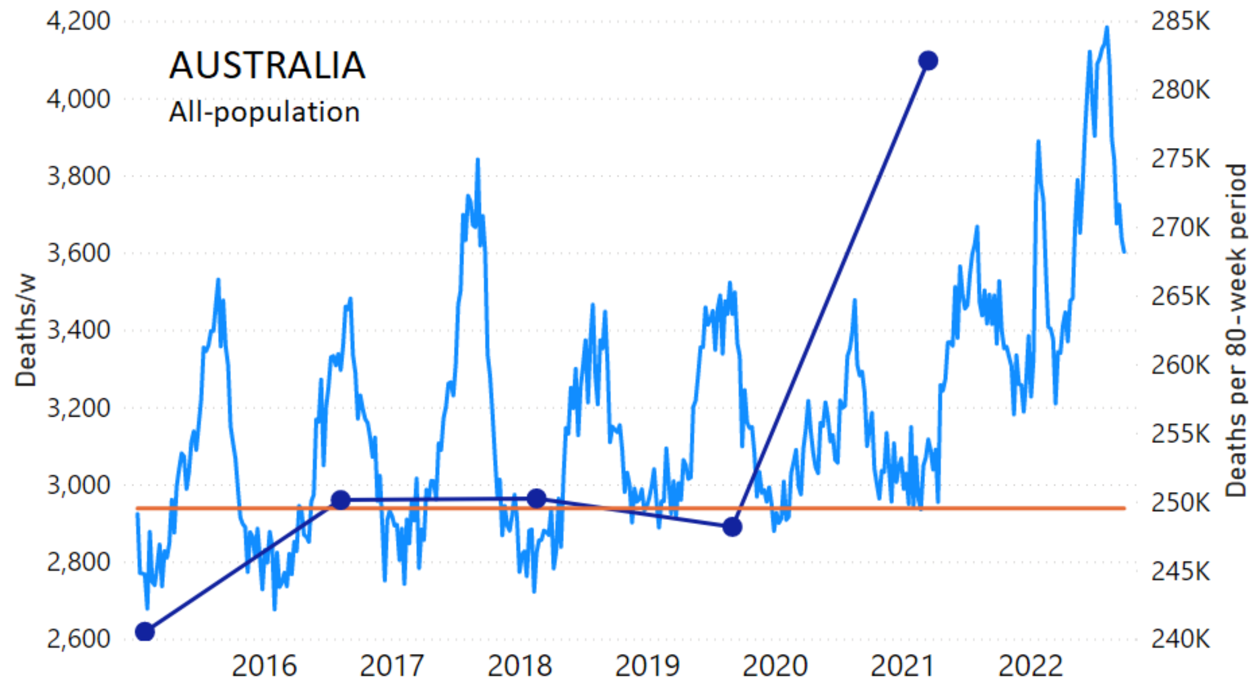
The same method can be adapted to any region of interest of sub-annual duration, by translating the window of integration (of the region of interest) backwards by increments of one year.

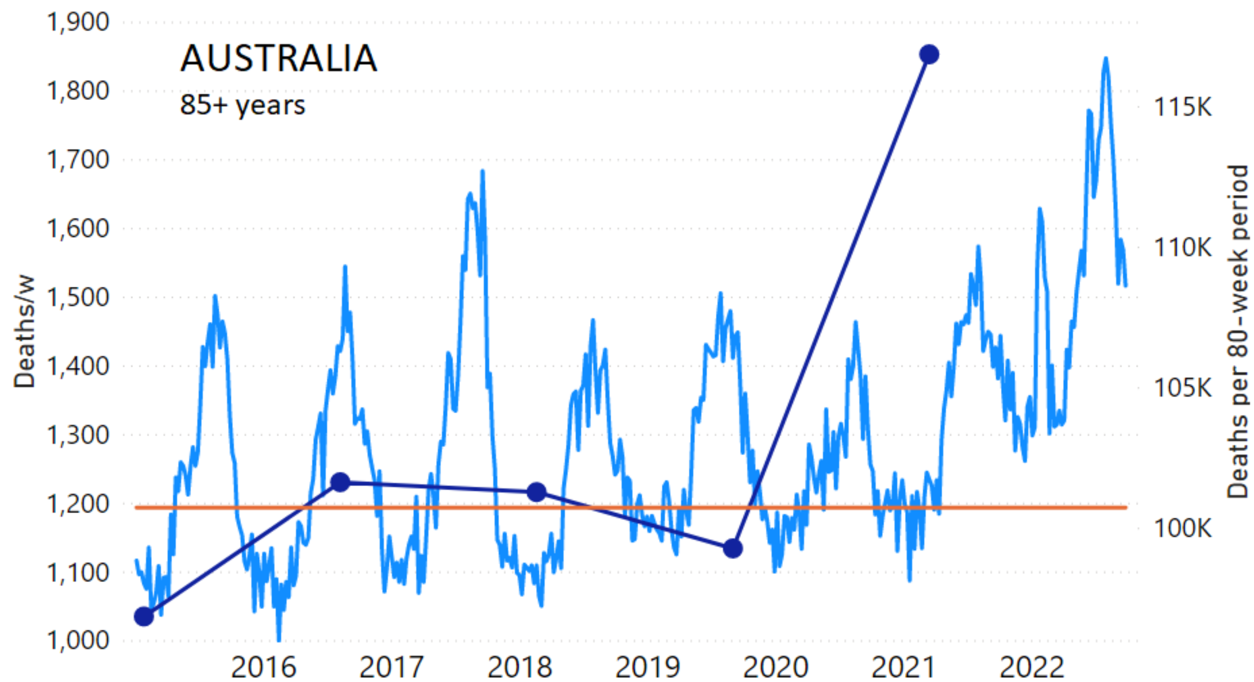
The above-described method is robust and ideally adapted to the nature of ACM data. Integrated ACM has a small statistical error. The large time-wise integration window removes difficulties arising from intrinsic seasonal variations. The historic trend is analysed without introducing any model assumptions or uncertainties beyond assuming that the near trend can be modelled by a straight line, where justified by the data itself. Such an analysis, for example, takes into account year to year changes in age-group cohort size arising from the age structure of the population. The only presumption is that a locally linear near trend for the unperturbed (ACM-wise unperturbed) population is realistic.

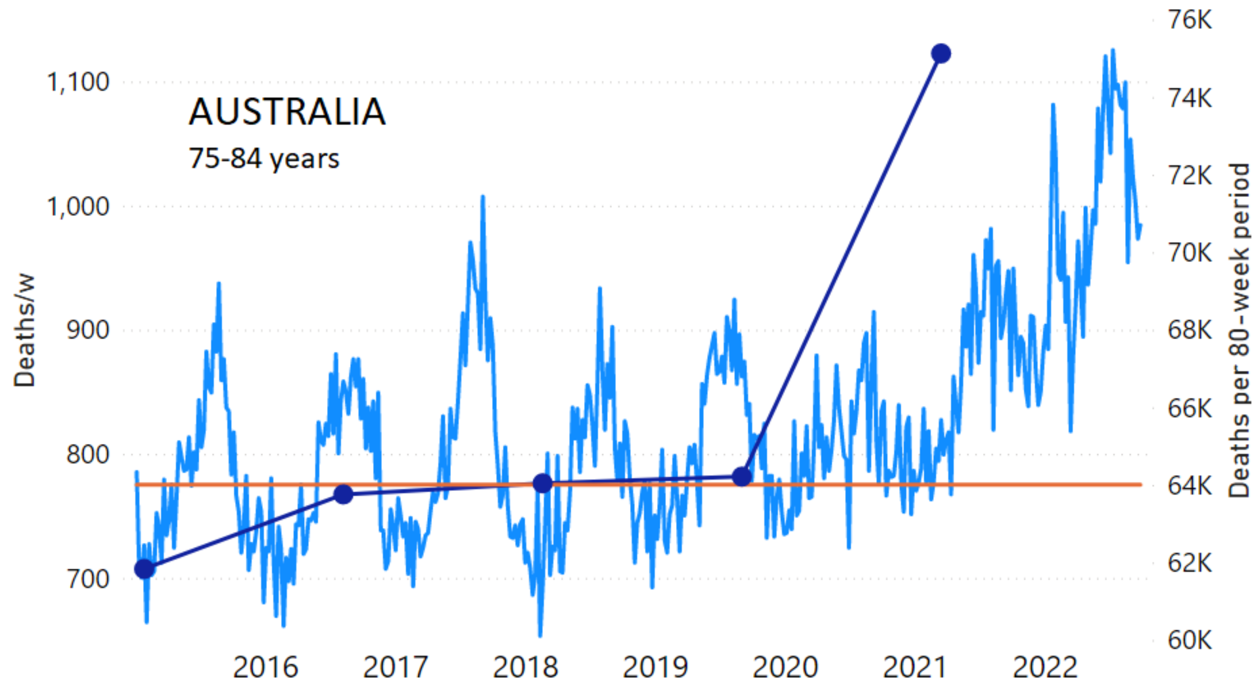
The calculation of the excess ACM by age group and for all-population for Australia is illustrated in Figure 1 (age groups as indicated in the figure), as follows. We used the three points sequentially preceding the vaccination period and imposed a horizontal line (zero slope of the fitted straight line), throughout (Figure 1).

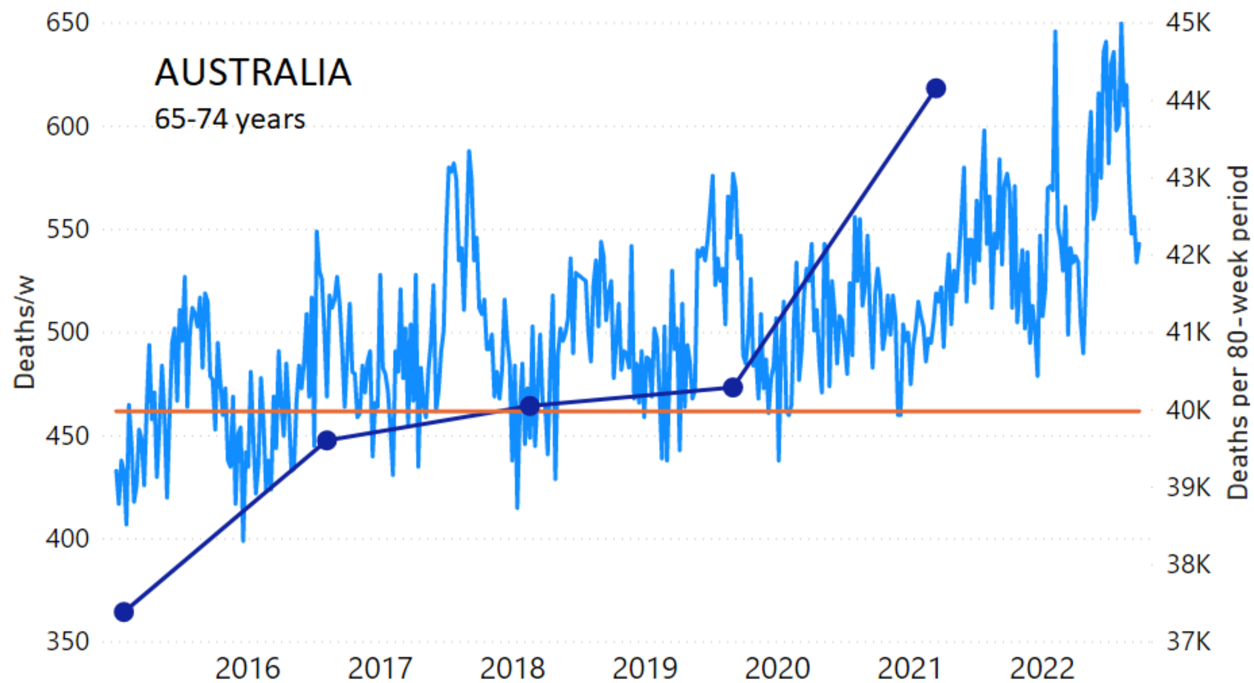
The details such as sources of official data, start and end points of integration, and methods for matching ACM and vaccine rollout data by age group, are provided in Appendix 1.

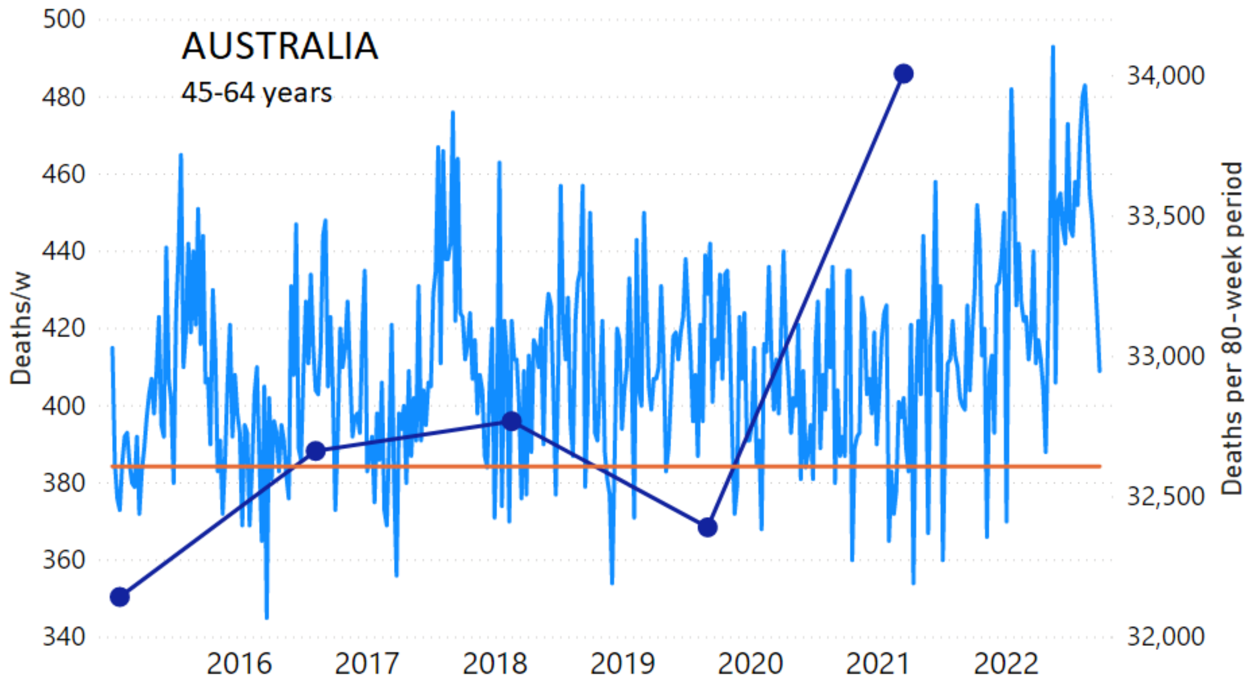
The integration period for Australia was fine-tuned and updated ACM data was implemented (see Appendix 1), compared to our previous analysis (Rancourt et al., 2022a), and the results are essentially identical.


AUSTRALIA
All-population
Deaths/w
Deaths per 80-week period
4,200
4,000
3,800
3,600
3,400
3,200
3,000
2,800
2,600
285K
280K
275K
270K
265K
260K
255K
250K
245K
240K
2016
2017
2018
2019
2020
2021
2022


AUSTRALIA
85+ years
Deaths/w
Deaths per 80-week period
1,900
1,800
1,700
1,600
1,500
1,400
1,300
1,200
1,100
1,000
115K
110K
105K
100K
2016
2017
2018
2019
2020
2021
2022


AUSTRALIA
75-84 years
Deaths/w
Deaths per 80-week period
1,100
1,000
900
800
700
76K
74K
72K
70K
68K
66K
64K
62K
60K
2016
2017
2018
2019
2020
2021
2022


AUSTRALIA
65-74 years
Deaths/w
Deaths per 80-week period
650
600
550
500
450
400
350
45K
44K
43K
42K
41K
40K
39K
38K
37K
2016
2017
2018
2019
2020
2021
2022



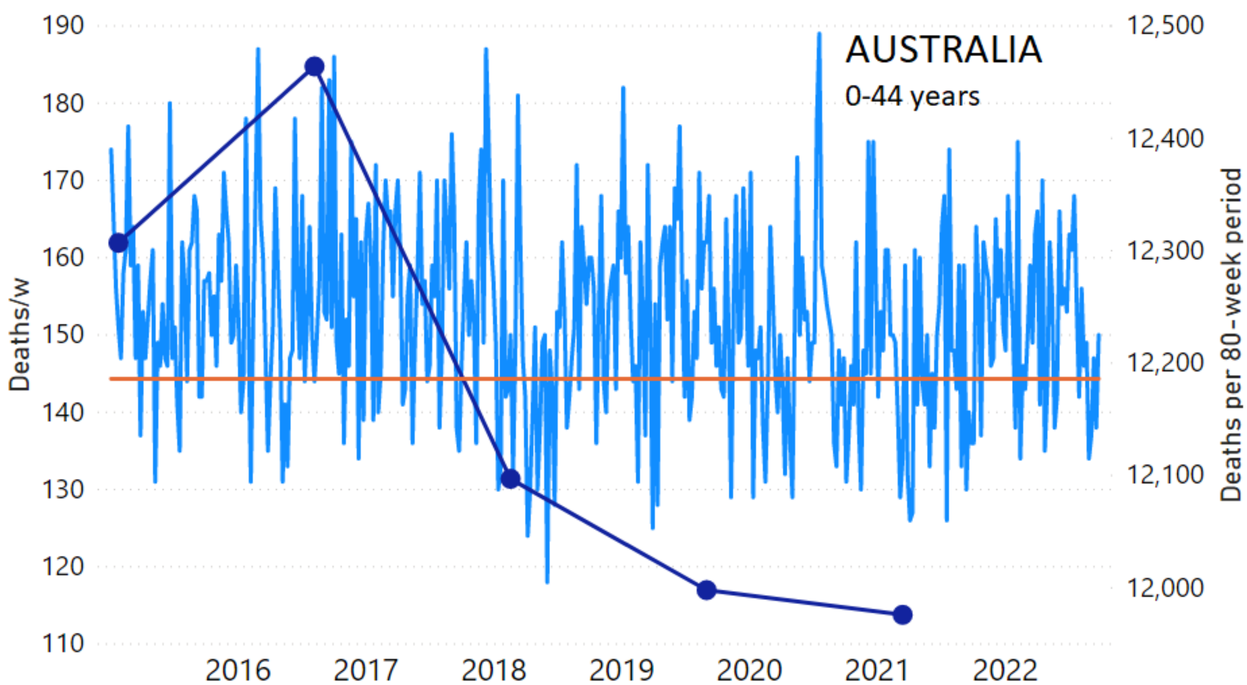


Figure 1: Australia, 2015-2022, by age group as indicated. ACM by week (light blue); integrated ACM by 80-week vaccination-period integration window (dark blue, points), the last point being for the actual vaccination period itself; extrapolation line used to calculate the excess ACM in the

vaccination period (orange). See the text for a description of the method, and Appendix 1 for details.

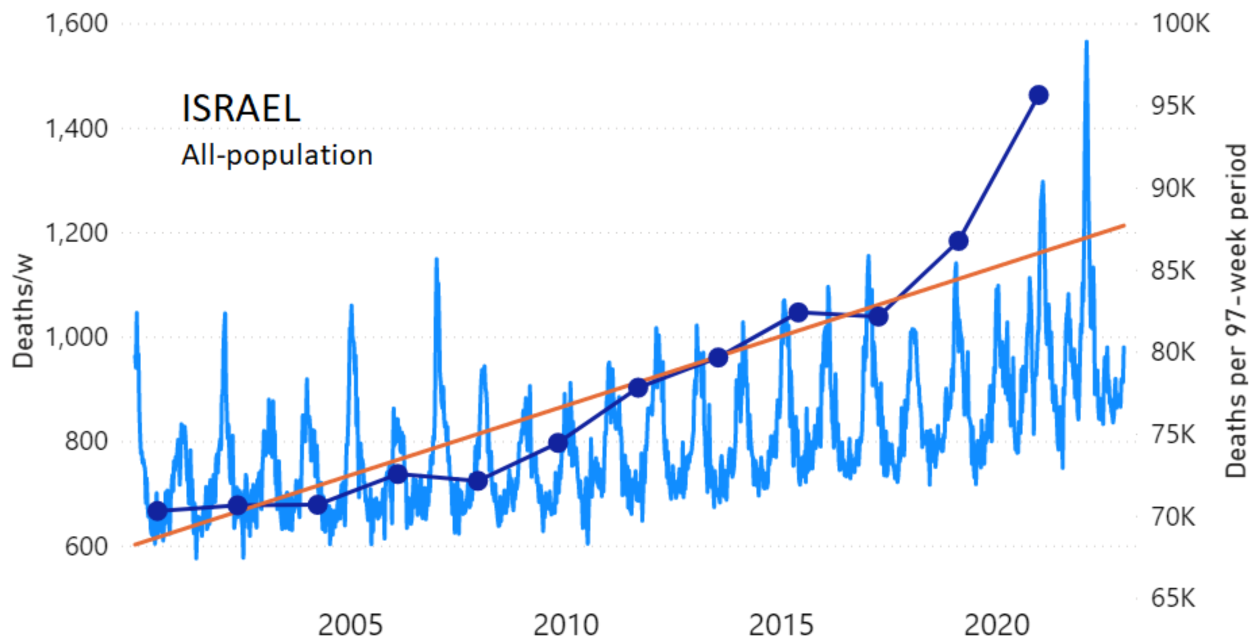
The youngest age group for Australia (0-44 years, Figure 1) shows our chosen extrapolation method not to be optimally suited to the ACM trend, however, in this age group the ACM is small, so this makes little difference. Furthermore, our method here automatically ensures that this difficulty is reflected in a larger estimated error, which is propagated to the calculated excess ACM.

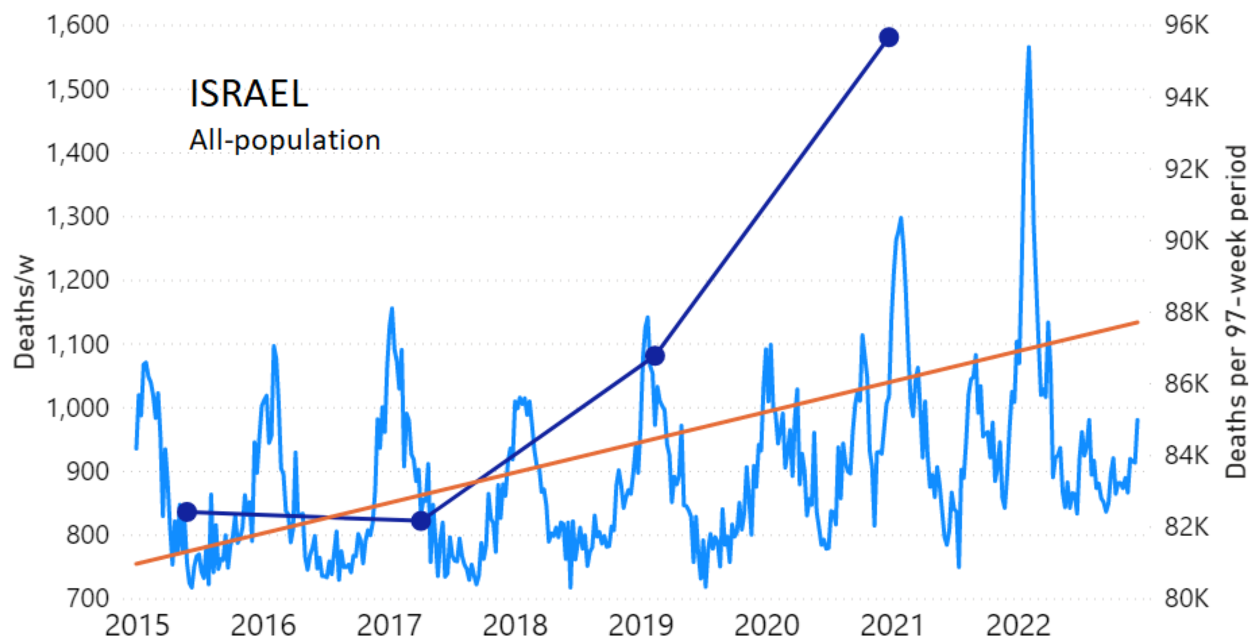
We do the same for Israel. The calculation of the excess ACM by age group and for all-population for Israel is illustrated in Figure 2 (age groups as indicated in the figure), as follows. Here we chose to use different sets of points to use in the extrapolation, as described in Appendix 1, and as can be surmised from Figure 2 itself.

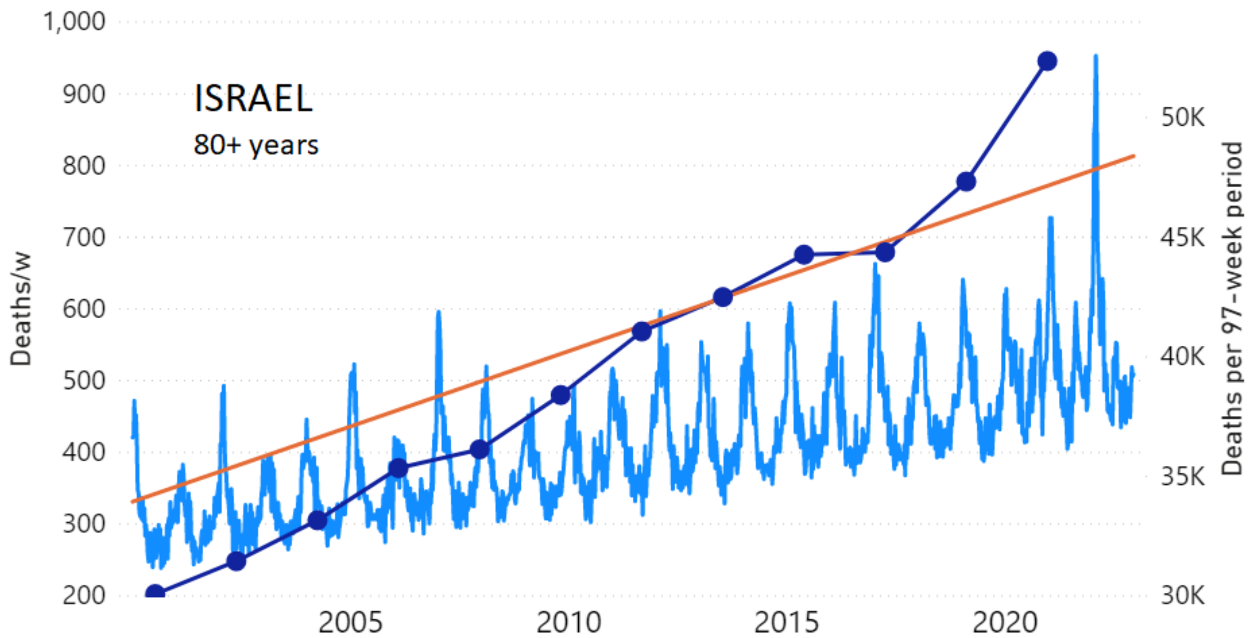
In this way, we account for the different historical trends in ACM that occur in the different age groups for Israel, and we avoid the point immediately preceding the vaccination period where it appears to include a significant excess mortality in the pre-vaccination period of the declared pandemic.

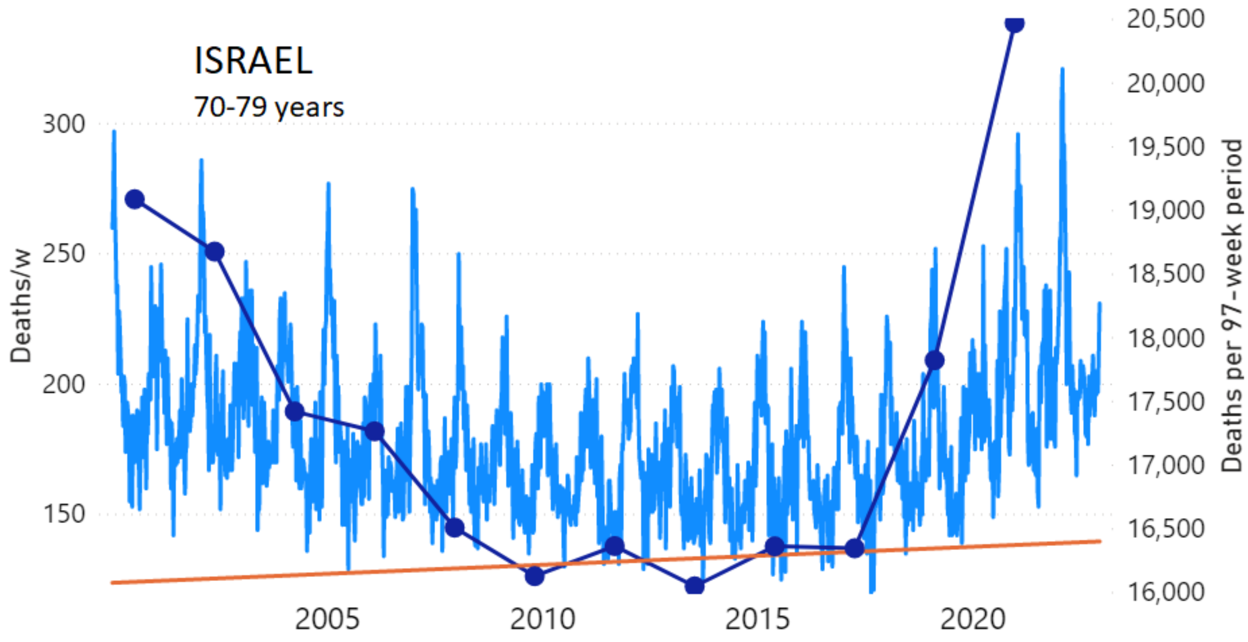
The details such as sources of official data, start and end points of integration, and methods for matching ACM and vaccine rollout data by age group, are provided in Appendix 1.

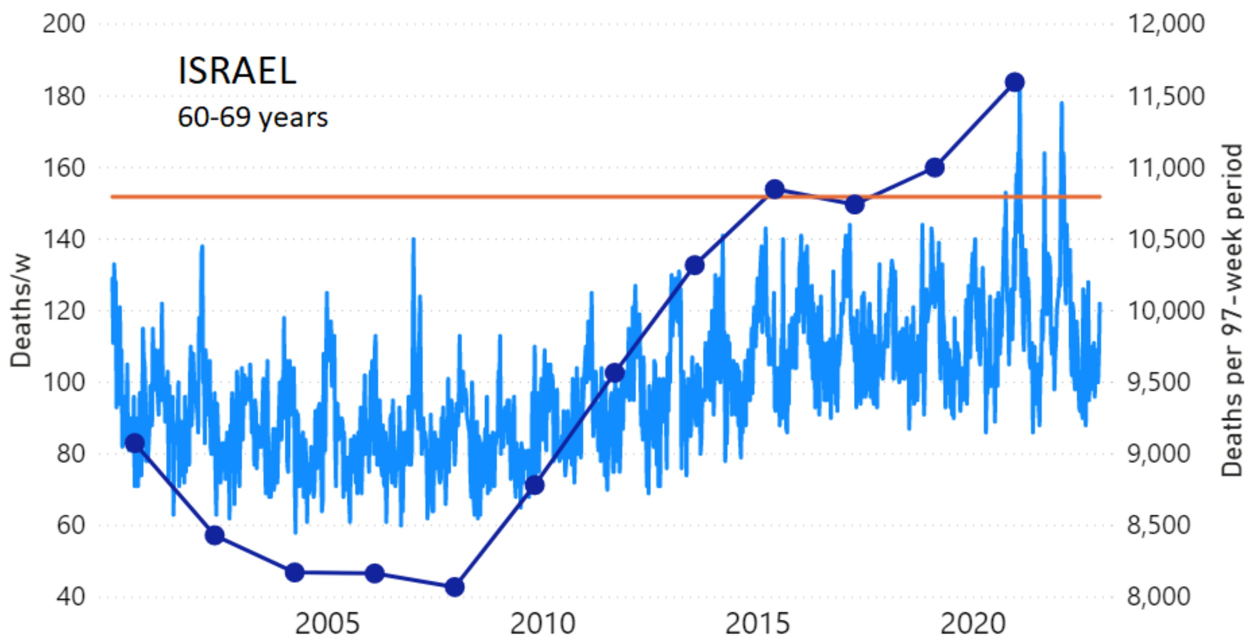
In terms of specific features in ACM by time, examples of synchronicity between ACM peaks and vaccine dose rollouts for Israel are shown in Appendix 2.

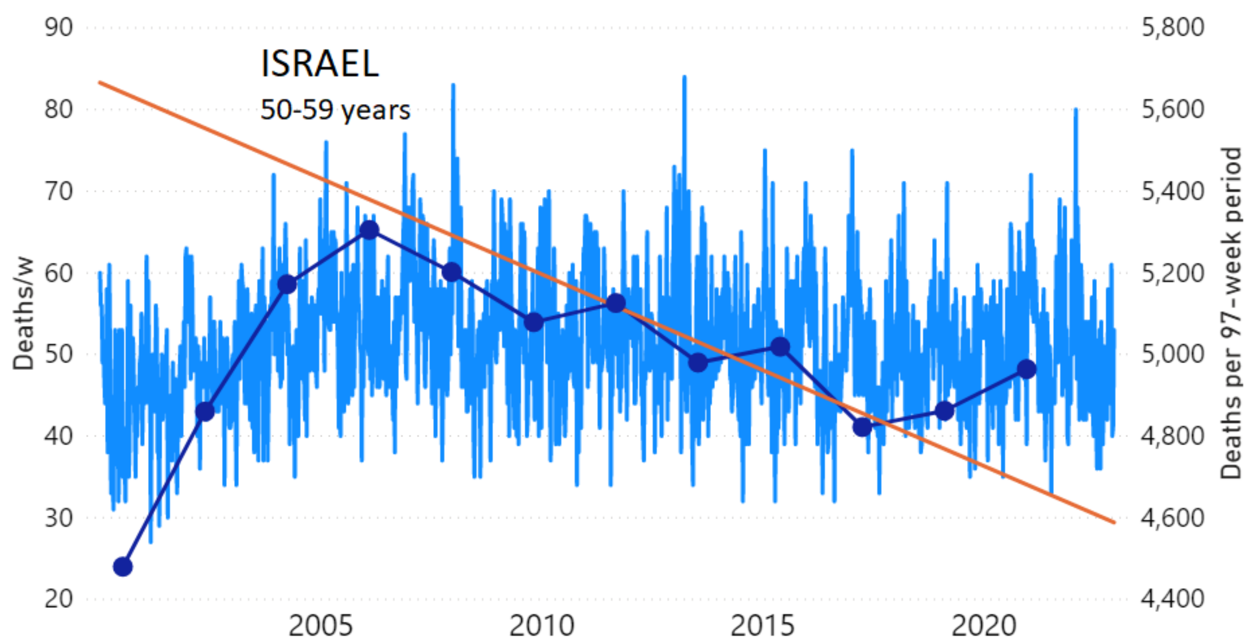

ISRAEL
All-population
Deaths/w
Deaths per 97-week period
1,600
1,400
1,200
1,000
800
600
100K
95K
90K
85K
80K
75K
70K
65K
2005
2010
2015
2020

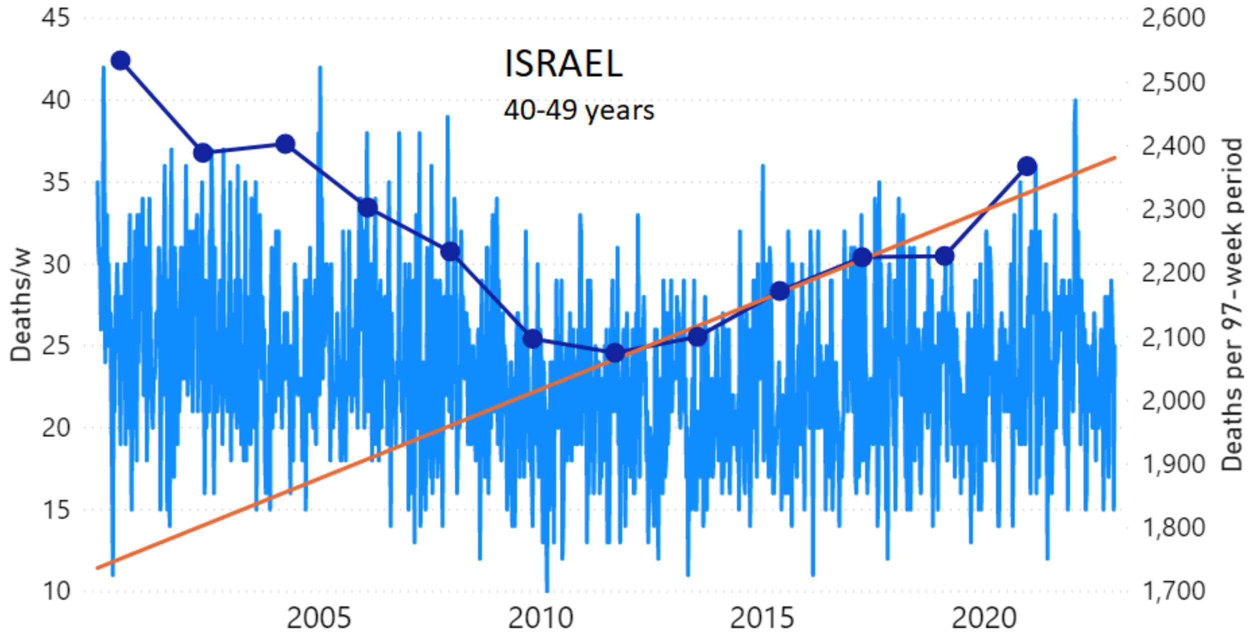

ISRAEL
All-population
Deaths/w
Deaths per 97-week period
1,600
1,500
1,400
1,300
1,200
1,100
1,000
900
800
700
96K
94K
92K
90K
88K
86K
84K
82K
80K
2015
2016
2017
2018
2019
2020
2021
2022

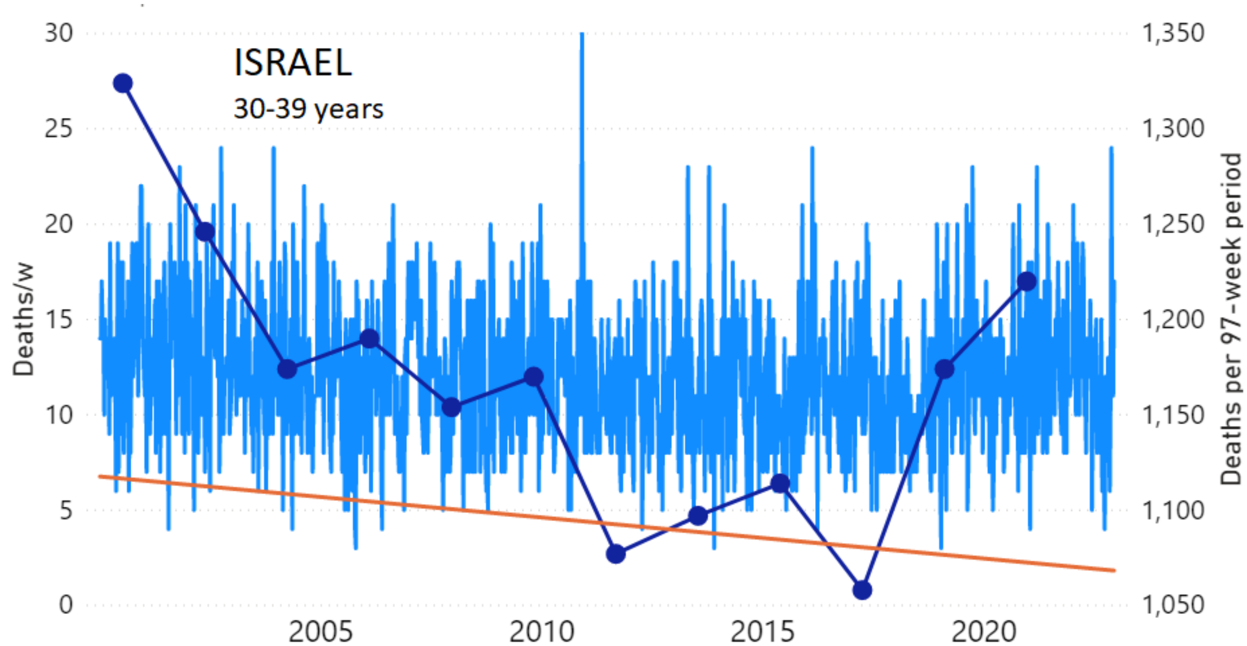

ISRAEL
80+ years
Deaths/w
1,000
900
800
700
600
500
400
300
200
2005
2010
2015
2020
50K
45K
40K
35K
30K
Deaths per 97-week period

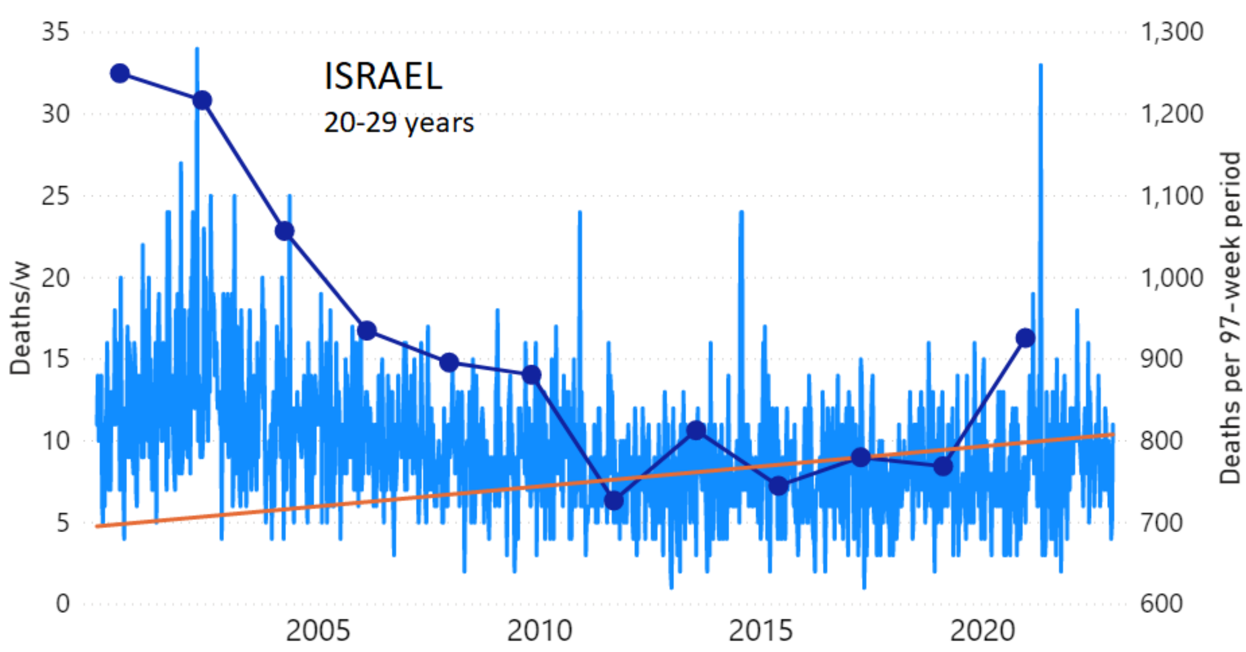

ISRAEL
70-79 years
Deaths/w
300
250
200
150
2005
2010
2015
2020
20,500
20,000
19,500
19,000
18,500
18,000
17,500
17,000
16,500
16,000
Deaths per 97-week period


ISRAEL
60-69 years
Deaths/w
Deaths per 97-week period
200
180
160
140
120
100
80
60
40
12,000
11,500
11,000
10,500
10,000
9,500
9,000
8,500
8,000
2005
2010
2015
2020


ISRAEL
50-59 years
Deaths/w
Deaths per 97-week period
90
80
70
60
50
40
30
20
5,800
5,600
5,400
5,200
5,000
4,800
4,600
4,400
2005
2010
2015
2020


ISRAEL
40-49 years
Deaths/w
Deaths per 97-week period
45
40
35
30
25
20
15
10
2,600
2,500
2,400
2,300
2,200
2,100
2,000
1,900
1,800
1,700
2005
2010
2015
2020


ISRAEL
30-39 years
Deaths/w
Deaths per 97-week period
30
25
20
15
10
5
0
1,350
1,300
1,250
1,200
1,150
1,100
1,050
2005
2010
2015
2020



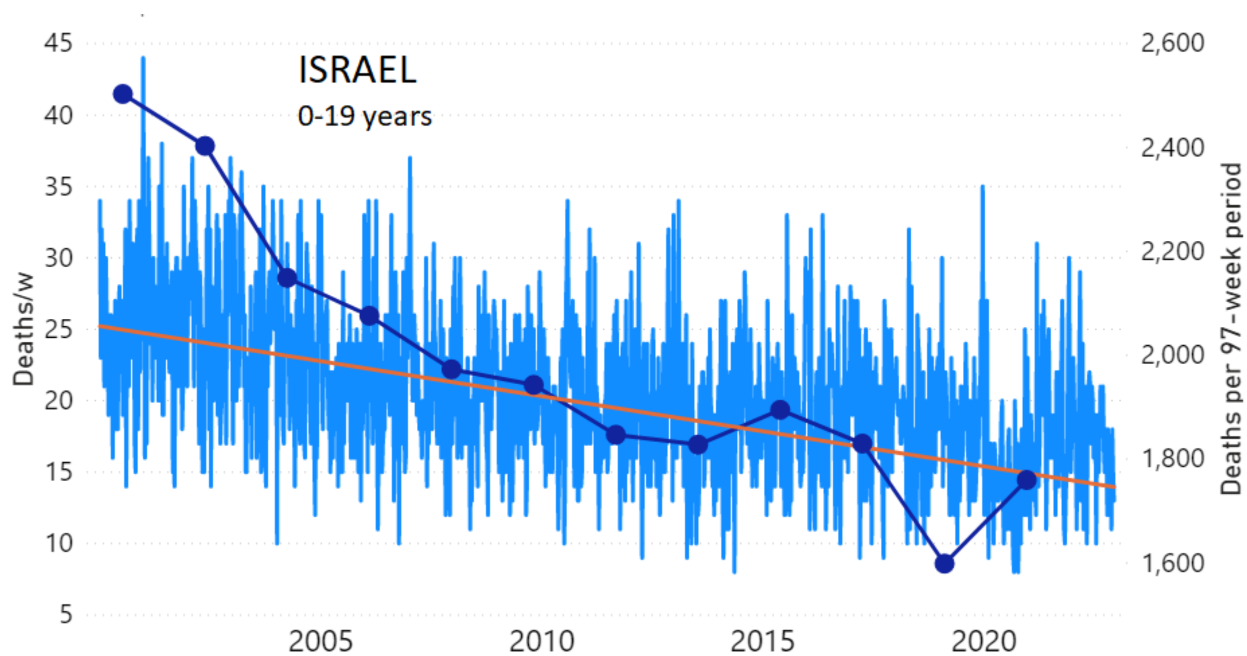


Figure 2: Israel, 2000-2022, by age group as indicated; and on expanded time axis 2015-2022 for all-population, as indicated. ACM by week (light blue); integrated ACM by 97-week vaccination-period integration window (dark blue, points), the last point being for the actual vaccination period itself; extrapolation line used to calculate the excess ACM in the vaccination period (orange). See the text for a description of the method, and Appendix 1 for details.

For Israel (Figure 2), although there is necessarily a degree of arbitrariness in the choice of the points to include in the linear regression, this does not significantly affect the results since:

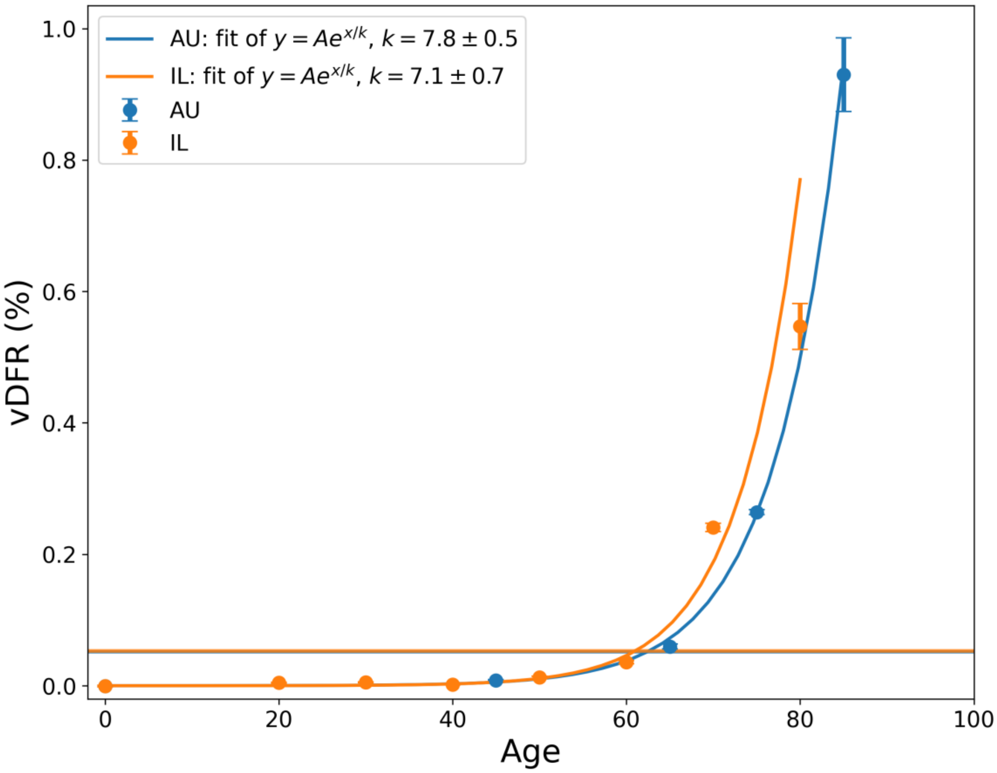
i. The effect (age-stratified excess ACM in the vaccination period) is large enough not to be sensitive to the said arbitrariness.
ii. The integrated ACM for the vaccination period is generally significantly and anomalously greater than its value for the immediately preceding integration period.
iii. Essentially the same result (age-stratified excess ACM in the vaccination period) occurs if we use the simplest possible method of taking the extrapolated vaccination-period ACM to be equal to the value for the immediately preceding point, which amounts to removing mortality occurring pre-vaccination in the pandemic period while assuming a locally constant trend in integrated ACM.

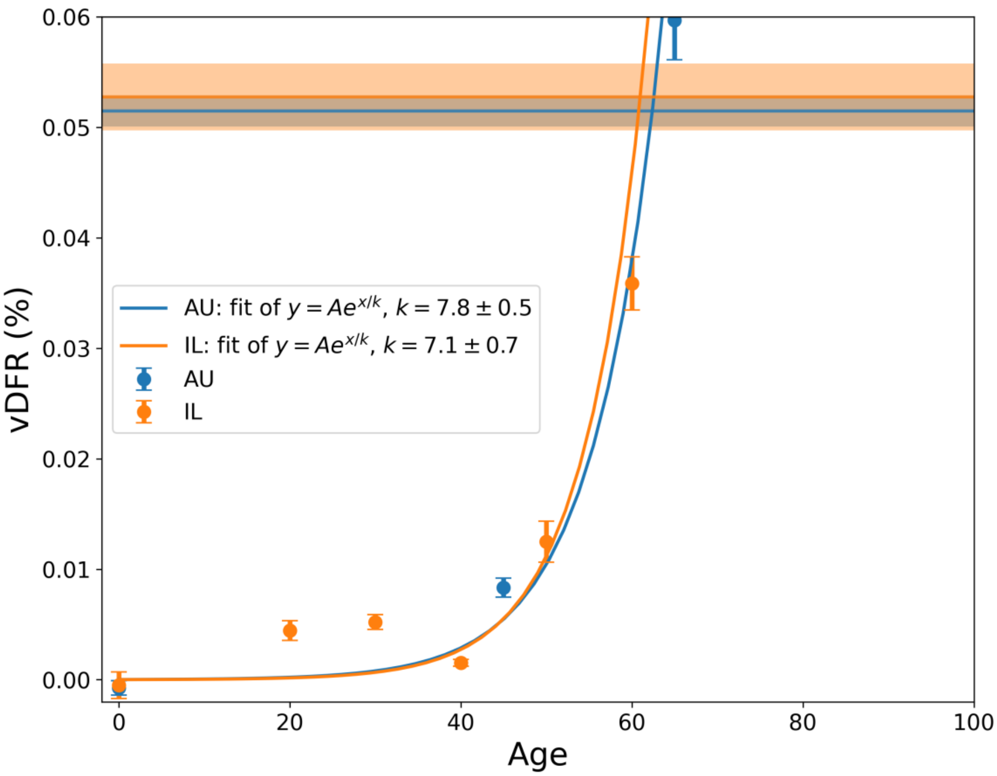
Tables 1 and 2 give the resulting age-stratified (and all-population) vDFR values for Australia and Israel, respectively. See Appendix 1 for details.

Table 1: AUSTRALIA			
Age group (years)	**Excess ACM in the vaccination period (±)**	**Vaccine doses in the vaccination period**	**vDFR (%) (±)**
All ages	32,610(890)	63,342,668	0.0515%(0.0014%)
85+	16,120(970)	1,734,308	0.930%(0.056%)
75-84	11,120(170)	4,210,402	0.264%(0.004%)
65-74	4,180(250)	6,994,831	0.0597%(0.0036%)
45-64	1,400(140)	16,791,268	0.00833%(0.00086%)
0-44	−210(190)	28,706,437	-0.00073%(0.00065%)

Table 2: ISRAEL			
Age group (years)	**Excess ACM in the vaccination period (±)**	**Vaccine doses in the vaccination period**	**vDFR (%) (±)**
All ages	9630(550)	18,251,720	0.0527%(0.0030%)
80+	5220(330)	954,235	0.547%(0.035%)
70-79	4100(110)	1,699,838	0.2410%(0.0065%)
60-69	800(54)	2,230,502	0.0359%(0.0024%)
50-59	283(42)	2,264,319	0.0125%(0.0019%)
40-49	42(8)	2,740,576	0.0015%(0.0003%)
30-39	148(19)	2,825,151	0.0052%(0.0007%)
20-29	128(26)	2,872,200	0.0045%(0.0009%)
0-19	−13(32)	2,664,899	−0.0005%(0.0012%)

The results from Tables 1 and 2 are plotted in Figure 3, with exponential fits, both on linear and logarithmic scales for vDFR.


AU: fit of $y = Ae^{x/k}$, $k = 7.8 \pm 0.5$
IL: fit of $y = Ae^{x/k}$, $k = 7.1 \pm 0.7$
AU
IL
vDFR (%)
Age
1.0
0.8
0.6
0.4
0.2
0.0
0
20
40
60
80
100


AU: fit of $y = Ae^{x/k}$, $k = 7.8 \pm 0.5$
IL: fit of $y = Ae^{x/k}$, $k = 7.1 \pm 0.7$
AU
IL
vDFR (%)
Age
0.06
0.05
0.04
0.03
0.02
0.01
0.00
0
20
40
60
80
100

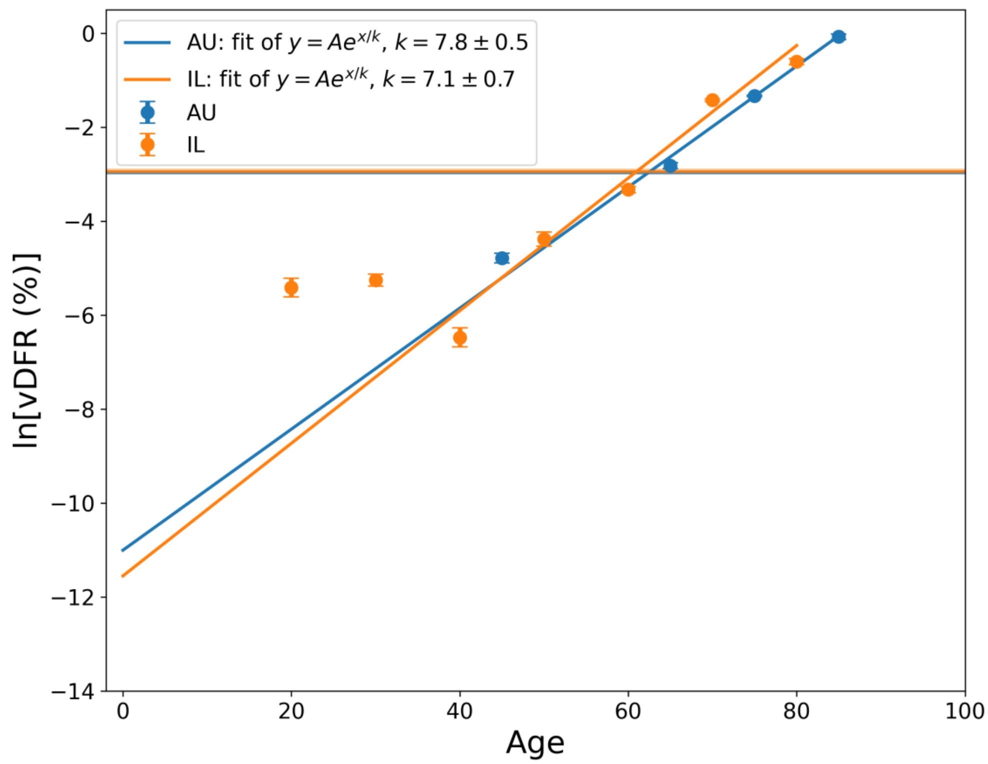


Figure 3: vDFR, which is the ratio of vaccine-induced deaths to vaccine doses delivered in the population of the specified age group, versus age for Israel (orange) and Australia (blue), on full (top) and expanded (middle) linear scales, and with semi-log scale (bottom). Horizontal bands are for the all-population values of vDFR. The age (X-axis value, years) assigned to a given age group is the starting age of the window of ages for the age group.

In Figure 3, the age (X-axis value, in years) assigned to a given age group is the starting age of the window of ages for the age group. This particular choice makes little difference because translating the x values by any constant number, for example, does not affect the doubling time obtained by fitting an exponential function, and only slightly affects the y intercept at x = 0 (the prefactor in the exponential).

The fitted exponentials (Figure 3) are of the form:

$$y = A \exp(x / k)$$

or

$$\text{vDFR} = A \exp(\text{Age} / k)$$

where A is the prefactor.

The doubling time (T2) is related to k as:

$$T2 = k\, \mathit{ln}(2).$$

The fitted values of k (and T2) are:

Fitted function: vDFR = A exp(Age / k)			
Country	**Number of points in fit**	**k (±) (years)**	**T2 (±) (years)**
AU	5	7.8(0.5)	5.4(0.3)
IL	8	7.1(0.7)	4.9(0.5)

This doubling time by age of approximately 5 years for risk of dying per injection of the COVID-19 vaccines is approximately half of the doubling time by age of 10 years for risk of dying per year of all causes in a modern human population, and of the main old-age diseases cancer, pneumonia and heart disease (Strekler and Mildvan, 1960). This implies a toxicity effect rather than simply inducing death by old age.

Furthermore, there is a non-exponential constant vDFR for young adults (vDFR ≈ 0.005 %, 20-40 years, Figure 3, Table 2). This suggests an accidental mechanism of death with a constant probability for these ages. One might postulate, for example, that vDFR is a product of a constant (age-independent) probability of accidental intra-vascular injection and a constant probability of death given intra-vascular injection. One might further postulate that one or both of these probabilities is larger in athletes with highly developed vascular systems and rapid circulatory rates (Cadegiani, 2022; Klein et al., 2022).

Our all-population value of vDFR of approximately 0.05 % (Figure 3, Tables 1 and 2) implies that in the USA, following the administration of approximately 670 million COVID-19 vaccine doses to date (669.60 million doses, up to 31 January 2023, *Our World in Data*),[2] approximately 330,000 USA residents would have died from the COVID-19 vaccines (1 in 1,000 on a population basis), assuming that elderly and vulnerable individuals are not more abundant or more aggressively targeted than in Australia or Israel. This number is comparable to the 278,000 fatalities found by Skidmore (2023) in his survey study for the USA. Our number of 330,000 is probably an underestimate, in light of the exponential dependence of vDFR with age that we have demonstrated and the known exceptionally large pools of highly vulnerable residents in the USA (Rancourt et al., 2022b).

Most importantly and concretely, our results establish a large vDFR in elderly people, as large as the 1 % measured for India when frail elderly people and patients with comorbidities were targeted (Rancourt, 2022), and when the same was presumably done in the high-poverty states of the USA, under the banner of vaccine equity programmes (Rancourt et al., 2022b).

The public health notion that elderly and vulnerable individuals must be prioritized for COVID-19 vaccination assumes:

i. a constant age-independent vDFR
ii. a small value of the vDFR optimistically estimated from managed trials, funded by the pharmaceutical industry

Our research shows that both assumptions (i and ii) are false, and far from reality in the field, on the scale of nations.

The said public health notion has always been baseless since it was not anchored in any sufficient evaluation of age-stratified risk of fatality from the injection (e.g., Veronese

[2] https://ourworldindata.org/grapher/cumulative-covid-vaccinations?country=~USA, consulted on 6 February 2023.

et al., 2021; Abbatecola et al., 2022; Gao et al., 2022), and is now proven to be contrary to reality. Prioritizing elderly people for vaccination, in the absence of relevant data, was reckless. Norway may be the only jurisdiction that immediately and publicly recognized a problem and changed its policy regarding vaccinating the most elderly and frail (*Reuters*, 18 January 2021; *Fortune*, 15 January 2021).

Some readers will be tempted to compare our results (Figure 3) with published age-stratified COVID-19 infection fatality rates (IFR) (e.g., COVID-19 Forecasting Team, 2022; Pezzullo et al., 2023). While in principle this is a correct approach of risk-benefit analysis, we believe that the IFR studies are not reliable, for the following reasons:

i. The deaths in the numerator of IFR are "COVID-19 deaths", and this cause of death assignation is susceptible to bias and is highly uncertain (e.g., Rancourt et al., 2022c; Rancourt et al., 2021).
ii. The number of infections, in the denominator of IFR, is reliant on molecular antibody tests, which are not specific and have not been sufficiently validated (e.g., Rancourt, 2021).
iii. If the IFR evaluations were valid, then it would be virtually impossible for jurisdictions like India and Australia to have no detectable excess ACM in the pre-vaccination period of the declared pandemic.
iv. We do not detect any excess ACM that can be attributed to COVID-19 in the jurisdictions that we have studied in detail (USA and all its states; Canada and its provinces; France and its departments and regions; Australia and its states).

The COVID-19 vaccines did not only not save lives but they are highly toxic.

On the global scale, given the 3.7 million fatalities in India alone, having vDFR = 1 % (Rancourt, 2022), and given the age-stratified vDFR results presented in this work, it is not unreasonable to assume an all-population global value of vDFR = 0.1 %. Based on the global number of COVID-19 vaccine doses administered to date (13.25 billion

doses, up to 24 January 2023, *Our World in Data*),[3] this would correspond to 13 million deaths from the COVID-19 vaccines worldwide. By comparison, the official World Health Organization (WHO) number of COVID-19 deaths to date is 6.8 million (6,817,478 deaths, reported to WHO, as 3 February 2023),[4] which are not detected as COVID-19 assignable deaths in ACM studies.

We are continuing our research on ACM, extending it to many national and sub-national jurisdictions. We hope that the present report will help put an end to the misguided and baseless public health policy that elderly people should be prioritized for vaccination.

(See Appendixes, below References)

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[3] https://ourworldindata.org/covid-vaccinations, as archived on 30 January 2023 here: https://archive.ph/u2gEO

[4] https://covid19.who.int/, as archived on 6 February 2023 here: https://archive.ph/boboE

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Appendix 1:
Data and Methods

Data

Table A1 describes the data used in this work and the sources of the data.

Data	Country	Period	Time unit	Filters	Source
ACM	Australia	2015-2022*	Week	Age group[1], sex	ABS, 2022
ACM	Israel	2000-2022**	Week	Age group[2], sex	CBS, 2022
Vaccines	Australia	2021-2023[+]	Week	Age group[3], sex	AG, 2022a AG, 2022b
Vaccines	Israel	2020-2022[++]	Day	Age group[4]	Data Gov, 2022
Population	Australia	2021	Year	Age group[5], sex	ABS, 2021

Table A1. Data retrieved. All-cause mortality (ACM), vaccine rollouts, population.

* At the date of access, data were available from week-1of 2015 (week finishing on January 4, 2015) to week-38 of 2022 (week finishing on September 25, 2022).
** At the date of access, data were available from week-1 of 2000 (week starting on January 3, 2000) to week-50 of 2022 (week starting on December 12, 2022).
[+] The reports of September 16, 2022 have been used in this work, reporting data as at September 14, 2022.
[++] At the date of access, data were available from Sunday December 20, 2020 to Tuesday October 25, 2022.
[1] 5 age groups: 0-44, 45-64, 65-74, 75-84, 85+
[2] 8 age groups: 0-19, 20-29, 30-39, 40-49, 50-59, 60-69, 70-79, 80+

[3] 19 age groups for vaccine doses 1 and 2: 5-11, 12-15, 16-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85-89, 90-94, 95+ (Excel file report, AG 2022a) and 14 age groups for vaccine doses 3 and 4: 5-11, 12-15, 16-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70+ (PDF file report, AG 2022b)
[4] 9 age groups: 0-19, 20-29, 30-39, 40-49, 50-59, 60-69, 70-79, 80-89, 90+
[5] 18 age groups: 0-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+

In addition to the data retrieved as per Table A1, we also examined cumulative vaccine dose by time data for Australia, as per our previous paper about Australia (Rancourt et al., 2022), from https://www.covid19data.com.au/vaccines.

In all the calculations and illustrations, both all-cause mortality (ACM, mortality from all causes of death) and numbers of vaccine doses administered are for the specific jurisdiction and age group.

Vaccine data for Australia are given as cumulative data (AG, 2022a and AG, 2022b).
Vaccine data for Israel are given as incremental data (Data Gov, 2022).
In the vaccines data of Israel, when the number of doses administered in a day is between 1 and 15, inclusively, the data shows "<15" (Data Gov, 2022). In order to have a figure to work with, we replaced "<15" by 15, choosing the upper bound of this unknown value. The net effect of this approximation is negligible.

For the vaccine data in Australia, doses 1 and doses 2 are given for 19 age groups (AG, 2022a), which cover the age groups of the ACM by age data (ABS, 2022). However, for doses 3 and 4, 14 age groups are given (AG, 2022b), which do not match the same age groups as for the ACM by age data (ABS, 2022). For this reason, we proceeded as follows.

Figure A1 is the figure from the Australian Government, on page 7 of their report (AG, 2022b):

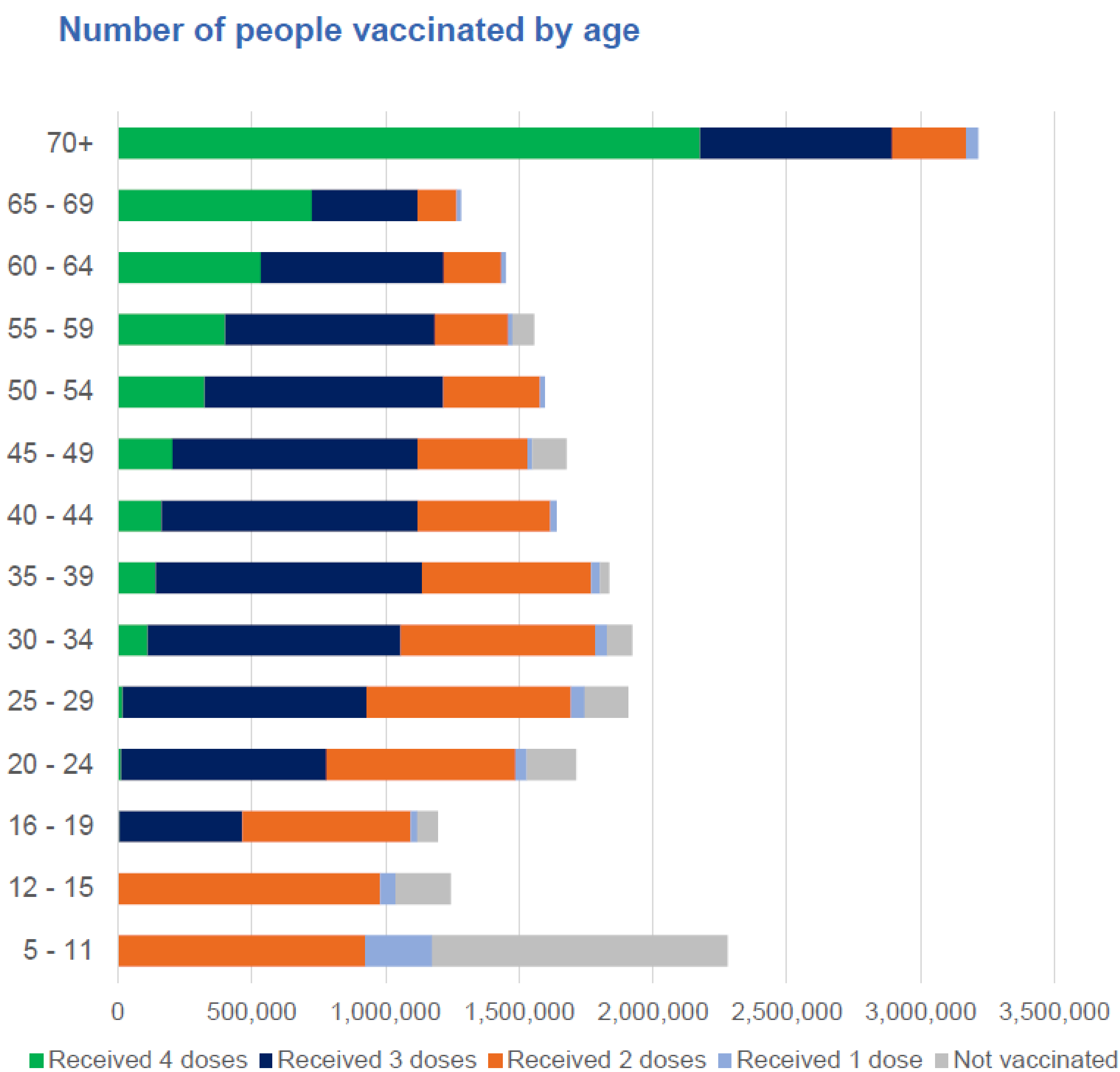


Figure A1. Vaccinations by age from the Australian Government, report of September 16, 2022, page 7 (AG, 2022b).

First, we estimate the number of doses 3+4 administered by age group from this figure (Figure A1). This is done in Table A2.

Age group	Measure (cm)	Doses 3+4
70+	8.40	2,896,551
65-69	3.23	1,113,793
60-64	3.51	1,210,344
55-59	3.41	1,175,862
50-54	3.53	1,217,241
45-49	3.25	1,120,689
40-44	3.25	1,120,689

35-39	3.3	1,137,931
30-34	3.1	1,068,965
25-29	2.7	931,034
20-24	2.25	775,862
16-19	1.33	458,620

Table A2. Estimation of the number of doses 3+4 by age group from AG, 2022b.
Scale used = 1,000,000 people for 2.9 cm.

Next, we estimate the number of doses 3+4 for the missing age groups: the 70-74, 75-84 and 85+ age groups. We assume and use a simple proportion of the population of those age groups (ABS, 2021). This is done in Table A3.

Age group	Population (ABS, 2021)	Doses 3+4
85+	542,342	510,100
75-84	1,376,518	1,294,687
70-74	1,160,768	1,091,762

Table A3. Estimation of the number of doses 3+4 for the 70-74, 75-84 and 85+ age groups.

Finally, we sum the estimations from Table A2 and Table A3 into relevant age groups to get the final number of doses 3+4 by ACM age group for Australia. This is done in Table A4.

Age group	Estimated number of doses 3+4
85+	510,100
75-84	1,294,687
65-74	2,205,555
45-64	4,724,136
0-44	5,493,101

Table A4. Estimation of the number of doses 3+4 by age group in Australia.

These age groups (Table A4) match those of the mortality data for Australia. Note that for the age group 0-44, doses 3 and 4 are for ages 16-44 years. There is no data for doses 3 and 4 for ages 0-15 years in Figure A1 (AG, 2022b).

Vaccination periods

For Israel, we use the same start date (week) of the vaccination period for all age groups. The integration of number of vaccine doses over the vaccine period is inclusive of the first and last weeks defining the said period. The same holds for integrated ACM periods.

For Australia, we use the vaccine-period end-date cumulative value of number of administered vaccine doses.

Table A5 defines the vaccination periods used in this work.

Country	Beginning	Ending	Duration (in weeks)
Australia	Week-10 of 2021	Week-37 of 2022	80
Israel	Week-52 of 2020	Week-43 of 2022	97

Table A5. Vaccination periods for Australia and Israel used in this work.
"The week number is based on the ISO (International Organization for Standardisation) week date system. In this system, weeks are defined as seven-day periods which start on a Monday. Week 1 of any given year is the week which starts on the Monday closest to 1 January, and for which the majority of its days fall in January (i.e. four days or more). Week 1 therefore always contains the 4th of January and always contains the first Thursday of the year. Using the ISO structure, some years (e.g. 2015 and 2020) contain 53 weeks." (definition from ABS, 2022).

Trendlines

Table A6 describes the method used to calculate the trendlines fitted to ACM integrated over the periods of equal duration as the duration of the vaccination period. The said trendlines are used to calculate the baseline integrated mortality in the vaccination period, in order to obtain the excess ACM of the vaccination period.

Country	Age group	Number of integration periods used*	Method
Australia	All	3	Average
Australia	85+	3	Average
Australia	75-84	3	Average
Australia	65-74	3	Average

Australia	45-64	3	Average
Australia	0-44	3	Average
Israel	All	4	Linear regression
Israel	80+	4	Linear regression
Israel	70-79	4	Linear regression
Israel	60-69	2	Average
Israel	50-59	4	Linear regression
Israel	40-49	4	Linear regression
Israel	30-39	4	Linear regression
Israel	20-29	4	Linear regression
Israel	0-19	6	Linear regression

Table A6. Method to estimate the trendlines. For Australia, we use the integrated ACM of the 3 periods prior to the vaccination period, each period being of duration equal to that of the vaccination period (80 weeks) and consecutive to each other, and we calculate the average. For Israel, we use the integrated ACM of the number of periods indicated in the table, prior to the first period directly preceding the vaccination period, each period being of duration equal to the duration of the vaccination period (97 weeks) and consecutive to each other, and we fit a linear trend.

***** This is the number of integrated ACM points (periods) used to calculate the trendlines.

The error in the calculated baseline value of integrated ACM over the vaccination period is estimated as the average of the absolute values of the residuals (fit to data) for the points (periods) used in the fit.

References for Appendix 1

ABS (2021): Australian Bureau of Statistics /// Population: Census - Information on sex and age /// accessed 30 January 2023 https://www.abs.gov.au/statistics/people/population/population-census/2021 — Note: The census that we used is for 2021, which was released in 2022.

ABS (2022): Australian Bureau of Statistics /// Provisional Mortality Statistics /// files "Provisional Mortality Statistics, Weekly Dashboard, Jan - Sep 2022" and "Deaths by week of occurrence, 2015-21" /// accessed 23 January 2023 https://www.abs.gov.au/statistics/health/causes-death/provisional-mortality-statistics/latest-release

AG (2022a): Australian Government /// COVID-19 vaccination – vaccination data – 16 September 2022 /// accessed 23 January 2023 https://www.health.gov.au/resources/publications/covid-19-vaccination-vaccination-data-16-september-2022?language=en

AG (2022b): Australian Government /// COVID-19 vaccine rollout update – 16 September 2022 /// accessed 30 January 2023 https://www.health.gov.au/resources/publications/covid-19-vaccine-rollout-update-16-september-2022?language=en

CBS (2022): Central Bureau of Statistics /// לוחות ותרשימים /// file “Death of Israeli residents, by week, gender, population group and age, 2000-2022” /// accessed 16 January 2023 https://www.cbs.gov.il/he/Pages/search/TableMaps.aspx?CbsSubject=%D7%AA%D7%9E%D7%95%D7%AA%D7%94%20%D7%95%D7%AA%D7%95%D7%97%D7%9C%D7%AA%20%D7%97%D7%99%D7%99%D7%9D

Data Gov (2022): Government databases /// גילאי המתחסנים /// accessed 29 December 2022 https://data.gov.il/dataset/covid-19/resource/57410611-936c-49a6-ac3c-838171055b1f

Rancourt et al. (2022): Rancourt, D.G., Baudin, M. and Mercier, J. /// Probable causal association between Australia’s new regime of high all-cause mortality and its COVID-19 vaccine rollout. /// *Correlation Research in the Public Interest*, 20 December 2022 /// https://correlation-canada.org/report-probable-causal-association-between-australias-new-regime-of-high-all-cause-mortality-and-its-covid-19-vaccine-rollout/

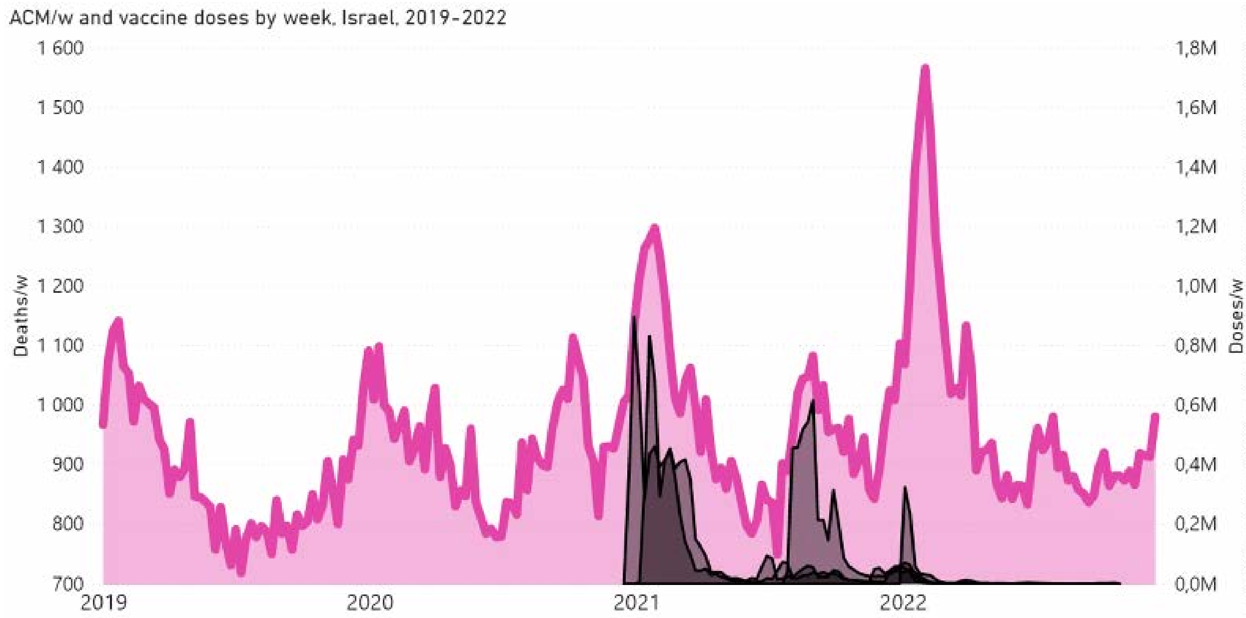
Appendix 2:
ACM and Vaccine Rollout Coincidences, for Israel, by Age Group

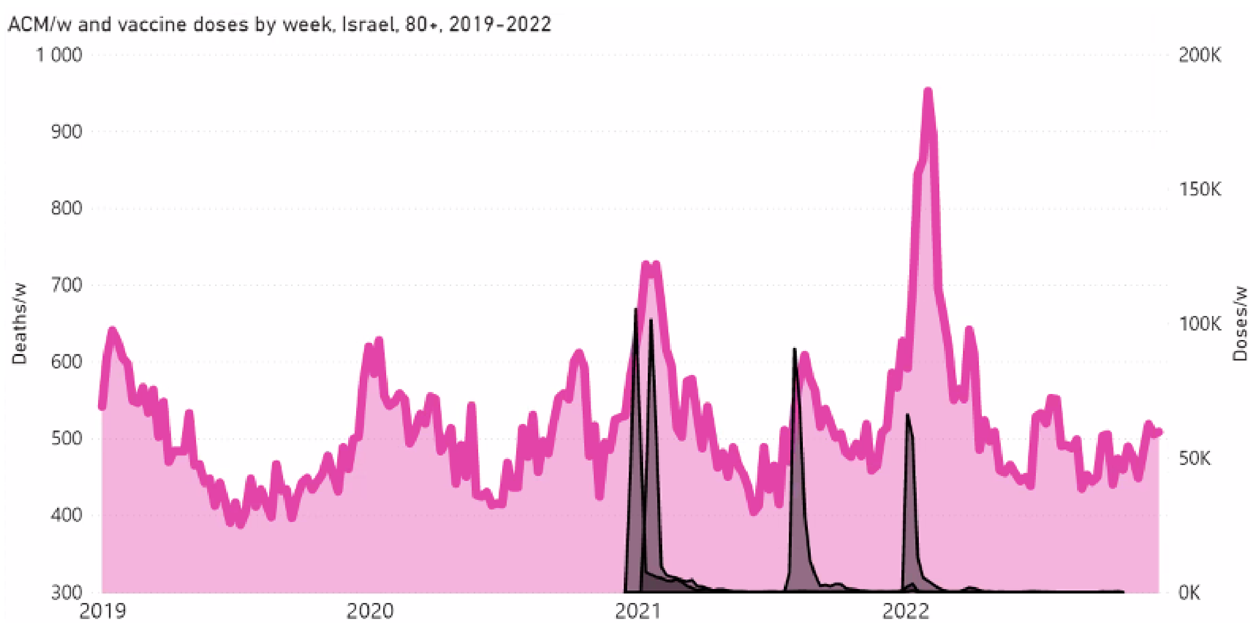
We have previously illustrated synchronicity between anomalous all-cause mortality (ACM) peaks and vaccine rollouts for:

- Australia (and each of its states New South Wales, Victoria, and Queensland),
- the USA (and its high-poverty states),
- the USA state of Michigan, and
- the Canadian province of Ontario

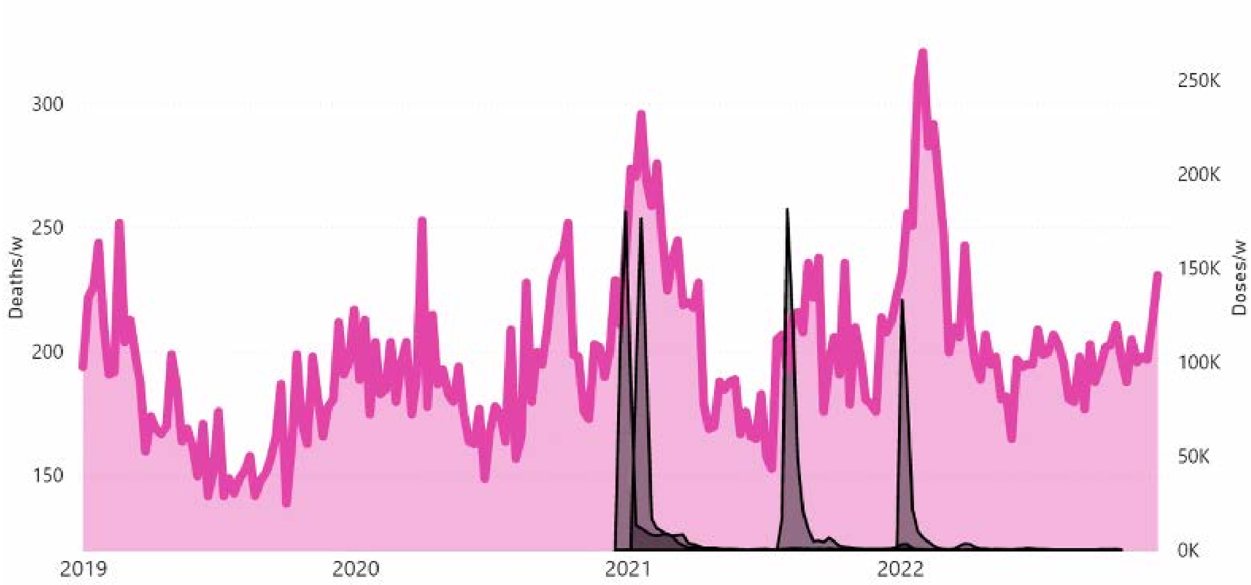
(See: Rancourt, D.G., Baudin, M. and Mercier, J. /// Probable causal association between Australia's new regime of high all-cause mortality and its COVID-19 vaccine rollout. /// *Correlation Research in the Public Interest*, 20 December 2022 /// https://correlation-canada.org/report-probable-causal-association-between-australias-new-regime-of-high-all-cause-mortality-and-its-covid-19-vaccine-rollout/)

Here, we examine this question for Israel and some of its age groups (as indicated), in the following Figure A2-F1:

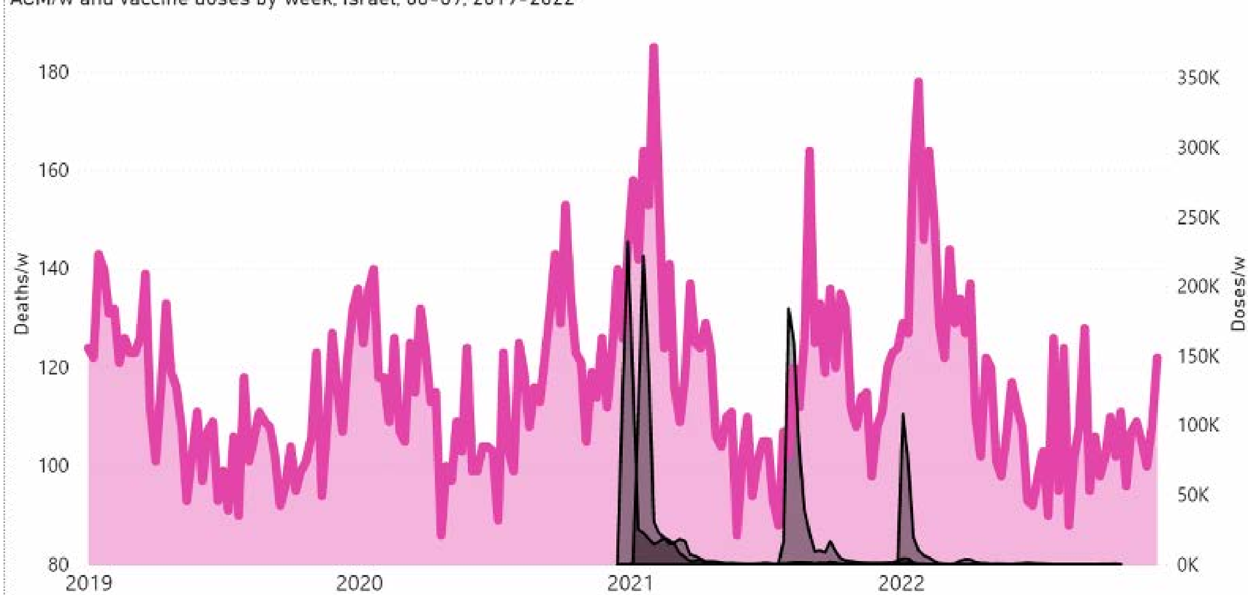

ACM/w and vaccine doses by week, Israel, 2019-2022
Deaths/w
Doses/w
1 600
1 500
1 400
1 300
1 200
1 100
1 000
900
800
700
1,8M
1,6M
1,4M
1,2M
1,0M
0,8M
0,6M
0,4M
0,2M
0,0M
2019
2020
2021
2022


ACM/w and vaccine doses by week, Israel, 80+, 2019-2022
Deaths/w
Doses/w
1 000
900
800
700
600
500
400
300
200K
150K
100K
50K
0K
2019
2020
2021
2022

ACM/w and vaccine doses by week, Israel, 70-79, 2019-2022



ACM/w and vaccine doses by week, Israel, 60-69, 2019-2022



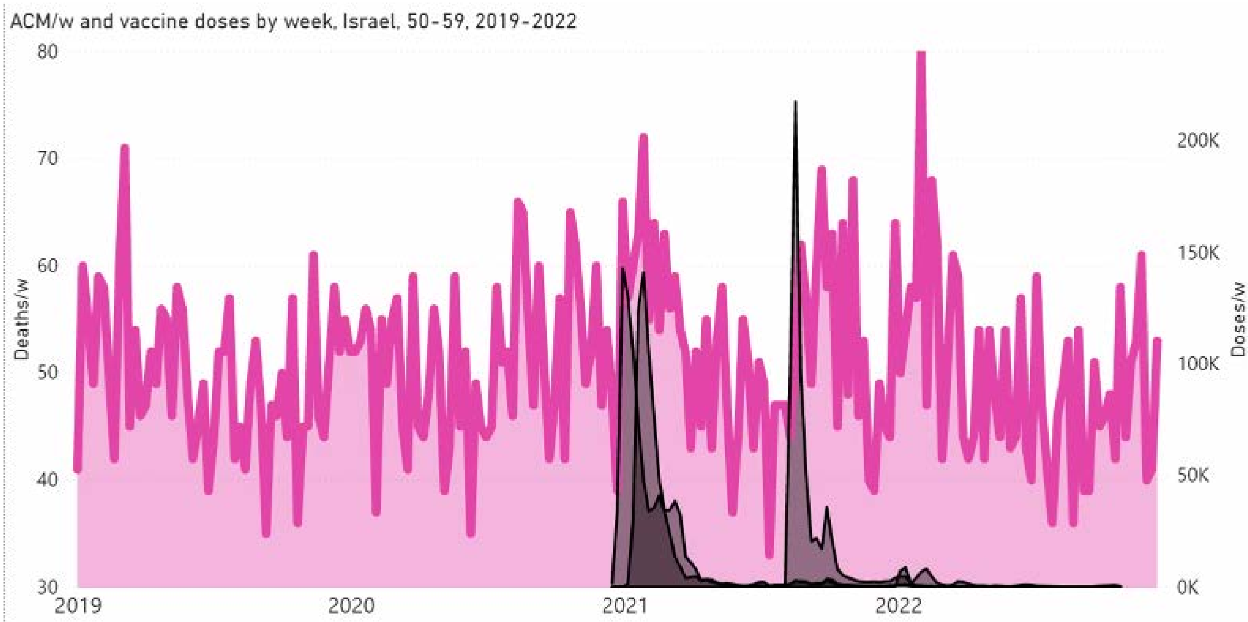


Figure A2 F1: Israel, 2019-2022, for (top to bottom, and as indicated) all ages, 80+ years, 70-79 years, 60-69 years, and 50-59 years. All-cause mortality (ACM) by week (pink, left y-scale); successive vaccine dose rollouts for doses 1, 2, 3 and 4, as numbers of doses administered by week (black and overlapping greys, right y-scale). The sources of all data are given in Appendix 1.

Predictions from standard epidemiological models of consequences of segregating and isolating vulnerable people into care facilities

Joseph Hickey, PhD[1,*] and Denis G. Rancourt, PhD[1]

[1] Correlation Research in the Public Interest (correlation-canada.org)

* Corresponding author: joseph.hickey@ucalgary.ca

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Abstract

Objectives: Since the declaration of the COVID-19 pandemic, many governments have imposed policies to reduce contacts between people who are presumed to be particularly vulnerable to dying from respiratory illnesses and the rest of the population. These policies typically address vulnerable individuals concentrated in centralized care facilities and entail limiting social contacts with visitors, staff members, and other care home residents. We use a standard epidemiological model to investigate the impact of such circumstances on the predicted infectious disease attack rates, for interacting robust and vulnerable populations.

Design: We implement a general susceptible-infectious-recovered (SIR) compartmental model with two populations: robust and vulnerable. The key model parameters are the per-individual frequencies of within-group (robust-robust and vulnerable-vulnerable) and between-group (robust-vulnerable and vulnerable-robust) infectious-susceptible contacts and the recovery times of individuals in the two groups, which can be significantly longer for vulnerable people.

Results: Across a large range of possible model parameters including degrees of segregation versus intermingling of vulnerable and robust individuals, we find that concentrating the most vulnerable into centralized care facilities virtually always increases the infectious disease attack rate in the vulnerable group, without significant benefit to the resistant group.

Conclusions: Isolated care homes of vulnerable residents are predicted to be the worst possible mixing circumstances for reducing harm in epidemic or pandemic conditions.

Strengths and limitations of this study

- We implement a simplest-possible sufficiently-realistic SIR model for an infectious respiratory disease with two interacting populations: robust and vulnerable.

- We investigate the predicted attack rates for a large range of parameters representing different degrees of segregation or isolation of the minority vulnerable population.
- We make broad-ranging conclusions about the consequences of segregation and isolation of vulnerable people, which apply to any epidemic model based on the SIR foundational assumptions.
- Large-parameter-range exploration is needed because the actual parameter values, especially the frequencies of infectious contacts, are not well delimited by empirical measurements and are often essentially unknown.

Introduction

During the COVID era (from the World Health Organization (WHO) 11 March 2020 COVID-19 pandemic declaration to present), many governments have imposed policies isolating or segregating people deemed highly vulnerable to respiratory disease, including by restricting movement into and out of long-term care homes where elderly and physically or mentally disabled people reside and reducing contacts between care home residents and staff (WHO, 2020a; WHO, 2020b, pp. 5, 22; WHO, 2020c, p. 10; Low et al., 2021).

Although it was known that isolation and loneliness can have serious negative health consequences for segregated vulnerable people (Armitage & Nellums, 2020; Holt-Lunstad et al., 2015; Valtorta et al., 2016), and although it was known that residents concentrated in care homes are particularly vulnerable to infectious diseases (Strausbaugh et al., 2003; Meyer, 2004; Monto et al., 2004; Gozalo et al., 2012; Lansbury et al., 2017), and although data from the spring of 2020 showed disproportionately large all-cause mortality increases in long-term care homes that were positively correlated with the number of care home residents (Amore et al., 2021; Sundaram et al., 2021), governments continued to implement policies confining vulnerable people into care homes and reducing social contacts with visitors and staff more than one year after the WHO's 11 March 2020 COVID-19 pandemic declaration.

Non-pharmaceutical interventions such as travel restrictions, workplace closures, and age-specific enforced social distancing or quarantining have been justified during the COVID era using theoretical infectious disease models based on the paradigm of spread by close-proximity pairwise contacts (Ferguson et al., 2020; Kreps & Kriner, 2020; Chang et al., 2020; Moss et al., 2020; Ogden et al., 2020). None of these models have been used to investigate the impact of segregation of the vulnerable into care homes.

Since policies isolating the vulnerable from contact with the majority of society have been widely applied, and since models are the main predictive tool used by governments to justify their public health policies, it is important to investigate model predictions for a large range of possible epidemiological parameters, rather than solely for limited ranges of parameters, which are not well constrained by empirical studies and which may be subject to political or institutional bias.

Large-range exploration of the parameters is needed because the actual parameter values are not well delimited by empirical measurements and are often essentially unknown; and because unexpected effects or magnitudes of effects can occur in different otherwise unexplored and relevant regions of the parameter space.

In order to appreciate the spectrum of outcomes that are possible in a given theoretical model, and its limitations and sensitivity to assumptions, it is crucial to base the model on the simplest-possible sufficiently realistic conceptual foundation and only add extensions incrementally (Garnett & Anderson, 1996; Siegenfeld et al., 2020). This approach optimizes relevance and minimizes confounding the results with complexity and intangible propagation of error. Focusing on only the core model ingredients limits the dimensionality of the model, permitting the needed examination of the model's outcomes across a comprehensive range of parameter values. To the extent that this approach is not adopted, the model becomes more removed from reality, because each additional complexity or sophisticated model element introduces new mechanisms, and therefore new assumptions about how those mechanisms function and new uncertainties about the values of their associated parameters.

At their core, the baseline epidemiological models on which essentially all more sophisticated models are built, have two main parameters determining whether an infectious disease epidemic emerges and, if it does, its magnitude and duration. These two parameters are: the rate at which individuals experience pairwise contacts with others that could result in transmission of the infection, and the rate at which infected individuals recover and become immune.

We construct a simple susceptible-infectious-recovered (SIR) epidemic model consisting of two interacting populations, one representing the relatively robust majority of society and the other the vulnerable minority. The different health states of individuals in the two populations are represented by their different recovery times upon infection, as is well established for respiratory diseases (Faes et al., 2020; Rhee et al., 2021). We investigate the size and duration of epidemics occurring for a broad range of different within- and between-population contact frequencies representing different segregation or isolation policy-linked behaviours. This approach allows us to make broad-ranging conclusions about the consequences of segregation of vulnerable people that apply to all epidemic models based on the SIR foundational assumptions.

Model

We implement a susceptible-infectious-recovered (SIR) model for two populations, indexed as population "*a*" and population "*b*". The total number of *a* individuals is N_a and the total number of *b* individuals is N_b.

Throughout this paper, we assign the *a* population to be the majority population of robust individuals, and the *b* population to be the minority population of vulnerable individuals.

Following the usual SIR model structure, a person can be in one of three states: susceptible to infection (S), infectious (I), or recovered and immune (R). If a susceptible person comes into contact with an infectious person, the susceptible person can become infectious, and infectious people eventually recover. The numbers of susceptible, infectious, and recovered people in group *i* at time *t* are therefore $S_i(t)$, $I_i(t)$, and $R_i(t)$, respectively, and $N_i = S_i(t) + I_i(t) + R_i(t)$.

The number of individuals in each of the three epidemiological compartments, in each of group *a* and *b*, evolve according to the following equations:

$$\frac{dS_a}{dt} = -S_a\left[c_{ab}\beta_{ab}\frac{I_b}{N_b} + c_{aa}\beta_{aa}\frac{I_a}{N_a}\right] \tag{1a}$$

$$\frac{dI_a}{dt} = S_a\left[c_{ab}\beta_{ab}\frac{I_b}{N_b} + c_{aa}\beta_{aa}\frac{I_a}{N_a}\right] - \gamma_a I_a \tag{1b}$$

$$\frac{dR_a}{dt} = \gamma_a I_a \tag{1c}$$

$$\frac{dS_b}{dt} = -S_b\left[c_{ba}\beta_{ba}\frac{I_a}{N_a} + c_{bb}\beta_{bb}\frac{I_b}{N_b}\right] \tag{1d}$$

$$\frac{dI_b}{dt} = S_b\left[c_{ba}\beta_{ba}\frac{I_a}{N_a} + c_{bb}\beta_{bb}\frac{I_b}{N_b}\right] - \gamma_b I_b \tag{1e}$$

$$\frac{dR_b}{dt} = \gamma_b I_b \tag{1f}$$

Equations 1a-f involve three sets of parameters, described below.

The parameters γ_a and γ_b represent the rates at which *a* and *b* individuals (robust and vulnerable individuals, respectively) recover from infection. Since the *b* population represents the minority, vulnerable population: $N_b \leq N_a$. Since they are more vulnerable than *a* individuals, *b* individuals take a longer time to recover from infection, such that $\gamma_b \leq \gamma_a$.

We use a value of γ_a = 75 yrs^{-1} corresponding to a recovery time of approximately 5 days for healthy individuals (Wolfel et al., 2020; CDC, 2022), and we consider three values of γ_b, equal to γ_a, $\gamma_a/2$, and $\gamma_a/4$, corresponding to recovery times of approximately 5, 10, and 20 days for the *b* individuals (Faes et al., 2020; Rhee et al., 2021).

The other two sets of parameters, c_{ij} and β_{ij}, are intrinsically dependent, such that one set is actually redundant, which can be understood as follows. β_{ij} represents the probability that a contact between a susceptible *i* (*a* or *b*) person and an infectious *j* person results in infection of the susceptible *i* person, whereas c_{ij} represents the frequency (number per unit time) of contacts between an *i* person and a *j* person. Therefore, we are free to make the following simplification. Without loss of generality, in this paper we set $\beta_{aa} = \beta_{bb} = \beta_{ab} = \beta_{ba} = 1$. This means that the only contacts considered and counted are by definition contacts that are guaranteed to result in transmission when the contact involves a susceptible *i* person and an infectious *j* person.

There is no reason or advantage to considering other definitions of c_{ij} having associated smaller values of β_{ij}; and it would make no difference in the calculated results arising from Eqns. 1a-f. Under this notational and conceptual simplification, the c_{ij} are the dominant control parameters in the model, along with the recovery rates γ_a and γ_b. We apply this interpretation of c_{ij} (arising from setting all the β parameters equal to 1) throughout the remainder of the paper.

The within-group contact frequencies, c_{aa} and c_{bb} are independent of one another. The between-group contact frequencies c_{ab} and c_{ba} are also independent. However, we impose the following relationship between c_{ab} and c_{ba}, modulated by the coefficient λ:

$$c_{ab} = \lambda \frac{c_{ba} N_b}{N_a} \tag{2}$$

A value of λ = 1 corresponds to a strict proportionality between c_{ab} and c_{ba} determined purely by the relative sizes of the populations of the two groups, as would be common to impose in the sliding definition of contact in which β_{ij} are undetermined (Garnett & Anderson, 1996).

In the present paper, λ = 1 effectively means that pairwise contact events that are of a physical proximity and duration sufficient to guarantee infection of a susceptible *b* person by an infectious *a* person are also sufficient to guarantee infection of a susceptible *a* person by an infectious *b* person. However, in principle, λ can take values less than 1, due to the more resistant health status of *a* individuals compared to *b* individuals. Since, given the relative sizes of the populations N_a and N_b, c_{ab} is much smaller than c_{ba} and typically much smaller than c_{aa} in our analyses, we use a value of λ = 1 in the main text of this paper. In the Appendices, we show that our results are robust against smaller values of λ.

We also define $c_a = c_{aa} + c_{ab}$ and $c_b = c_{bb} + c_{ba}$ to be the total contact frequencies of *a* and *b* people, respectively. The majority, robust (*a*) population is typically younger and more socially active than the minority, vulnerable (*b*) population, such that the frequency of all person-to-person contacts is generally higher in the *a* group than the *b* group (Prem et al., 2017). However, when c_a and c_b represent the frequency of only those types of contacts that are guaranteed to result in infection of a susceptible individual (as per our simplifying assumption that $\beta_{aa} = \beta_{bb} = \beta_{ab} = \beta_{ba} = 1$, in the present article), then it is not unreasonable to consider that c_b can be greater or significantly greater than c_a, due to the frailer health status of the *b* individuals.

Results

We examine the epidemic outcomes for the robust (*a*) and vulnerable (*b*) populations for a large range of possible contact frequencies and recovery rates. For specificity, we use a total population of $N = 10^7$ individuals, with $N_a/N = P_a = 0.95$, such that the *a* population constitutes 95% of the entire society, and the *b* population 5%. The simulations are "seeded" with 100 infectious individuals inserted proportionally into each of the two groups, such that $I_a(t{=}0) = 95$ and $I_b(t{=}0) = 5$.

We verified that the results are the same on varying P_a, λ, and seeding magnitude and distribution, which is shown in the Appendices.

We define the attack rate among population *i* as the proportion of initially-susceptible *i* people who become infected during the epidemic:

$$A_i = \left(S_i(t_0) - S_i(t_f)\right)/S_i(t_0), \tag{3}$$

where $S_i(t_0)$ is the number of susceptible *i* people at the beginning of the epidemic and $S_i(t_f)$ is the number of susceptible *i* people remaining once there are no longer any infectious people in either of the two groups (*a* or *b*).

In order to examine the impact of policies that isolate or segregate the *b* individuals from the *a* group, we introduce the index *x* equal to the share of a *b* individual's contacts that are with *a* people:

$$x = c_{ba} / c_b, \tag{4}$$

When $x = 0$, *b* individuals only ever have contacts with other *b* individuals, and when $x = 1$, *b* individuals only ever have contacts with *a* people. In this way, *x*, represents the degree of segregation versus intermingling of the *a* and *b* groups. Complete segregation is $x = 0$. Complete *a*-*b* intermingling, while avoiding all *b*-*b* contacts, is $x = 1$.

Fig. 1 shows the evolution of the epidemic (number of new cases per day, over time) in the *a* and *b* groups, for different values of *x*. In this example, c_a is slightly larger than γ_a (in order that c_a / γ_a ("R_0") ≈ 1.1 > 1 such that an epidemic would occur in the *a* group if it were completely isolated from the *b* group) and c_b is 25% larger than c_a. $\gamma_b = \gamma_a/4$, such that *b* people take four times as long to recover from infection as *a* people.

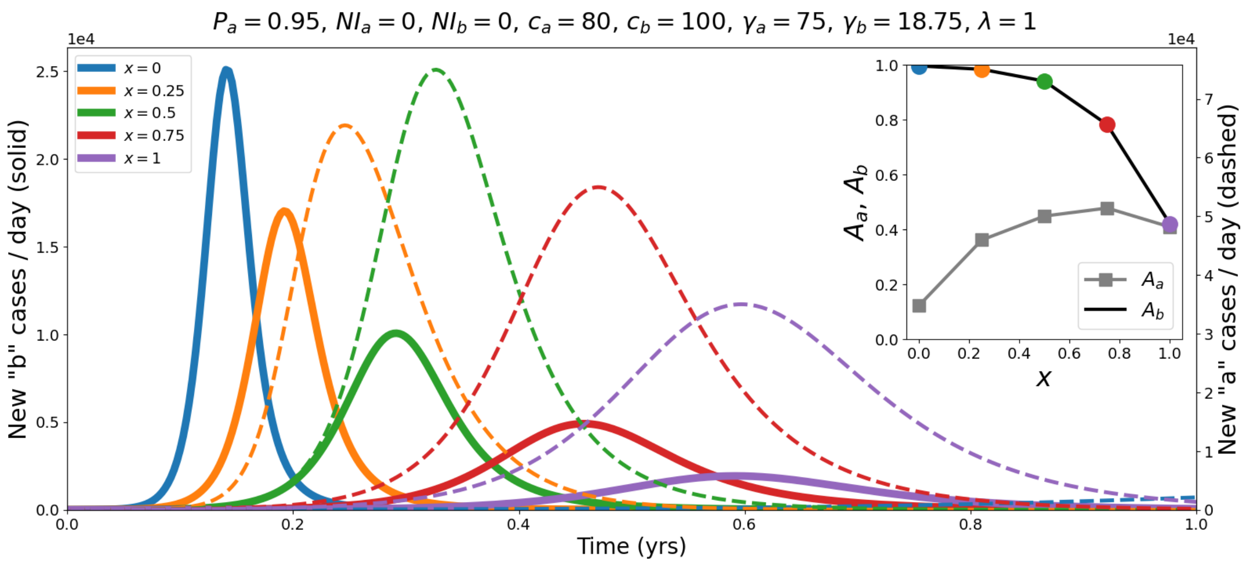


Figure 1: Epidemic curves showing the number of new cases per day in population *b* (vulnerable, minority group, solid lines, left y-axis) population *a* (robust, majority group, dashed lines, right y-axis), for different values of *x*, and for the fixed model parameters indicated above the figure. Inset: attack-rates A_a and A_b as functions of *x* (coloured circles indicate the *x* values listed in the main figure legend).

As can be seen in Fig. 1, *x* (the degree of separation or intermingling) has a large effect on the size and duration of the epidemics occurring in both the *a* (robust, majority) and *b* (vulnerable, minority) groups.

When $x = 0$, *b* individuals only ever come into contact with other *b*'s, and the number of new cases per day in the *b* group rapidly surges, peaks, and decays, and essentially all of the *b* population becomes infected ($A_b \approx 1$, inset of Fig. 1). An epidemic also occurs in the *a* group, but the attack rate is smaller (A_a,

inset) and it takes significantly longer for the epidemic to transpire (see the dashed blue line in the extreme lower-right corner of Fig. 1).

In Fig. 1, as x is increased above 0, a larger and larger share of b contacts are with a individuals. In the b group, the epidemic size (peak value of new cases per day and attack-rate) decreases with increasing x and the duration of the epidemic increases. Going from x = 0.5 to x = 0.75 and x = 1, A_b is significantly decreased, to the point where less than half of the susceptible, vulnerable b population becomes infected. On the other hand, increasing x above 0 initially increases A_a and significantly shortens the time it takes for the number of new a cases per day to surge and decay. When x = 1, the epidemic curves for the a and b populations have their peaks at approximately the same time, and the attack rates become similar for the two groups.

Fig. 1 illustrates the important effect of x on the epidemic outcomes in the two populations. In particular, it is apparent that larger x (more contacts with robust individuals) can produce significantly better (lower attack rate) results for the minority vulnerable population. This is important if it is a general feature because the vulnerable individuals in the real world have higher risk of dying on being infected (COVID-19 Forecasting Team, 2022), which is the motivation for wanting to protect them.

Next, we present figures showing results across our large range of possible and reasonable c_a and c_b values, for different degrees of segregation vs. intermingling, x, between the a and b groups, and for the different values of γ_b representing different degrees of vulnerability of the b population.

Fig. 2 contains a collection of panels showing how the attack-rates A_a and A_b change as c_a and c_b are varied. Each panel corresponds to a choice of x and γ_b.

The panel in the upper-left corner of Fig. 2 corresponds to x = 0 and $\gamma_b = \gamma_a/4 = 18.75$. Since x = 0, there is complete segregation between the a and b groups. In this case, an epidemic emerges in the a group when $c_a > \gamma_a$ and in the b group when $c_b > \gamma_b$, and this can be seen by the fact that $A_a > 0$ when $c_a > 75$, for all values of c_b, and $A_b > 0$ when $c_b > 18.75$, for all values of c_a. Thus, when x = 0, we see the usual transition to an epidemic, which occurs in a one-population SIR model when $R = c/\gamma > 1$, in each group.

The panels in the second through fifth rows of Fig. 2 correspond to $x > 0$, progressively increasing up to x = 1 (fifth row). For many values of c_a, increasing x results in a shift upwards (to higher c_b values) of the red contour lines, indicating a decrease in A_b for fixed c_b.

For example, when γ_b = 18.75 (left column of panels), c_a = 20 and c_b = 40, the attack rate A_b is large when x = 0. However, as x is increased, the red contour lines shift upward, indicating a lowering of the attack rate at (c_a, c_b) = (20, 40), until A_b = 0 (no epidemic in the b population) in the second-last and last panels in the column (x = 0.75 and x = 1).

The positioning of the blue contour lines (A_a) is generally less affected by changes in x than that of the red contours. This is particularly evident for the case of $\gamma_b = \gamma_a$ (right column of panels). This is due to the asymmetry in the sizes of the populations of the a and b groups (N_b being 5% of the total population).

To better appreciate the model results summarized in the contour maps of Fig. 2, it is helpful to simultaneously examine the attack rates for a particular point in the (c_a, c_b) parameter-space as x is varied. This is shown in Figs. 3-5.

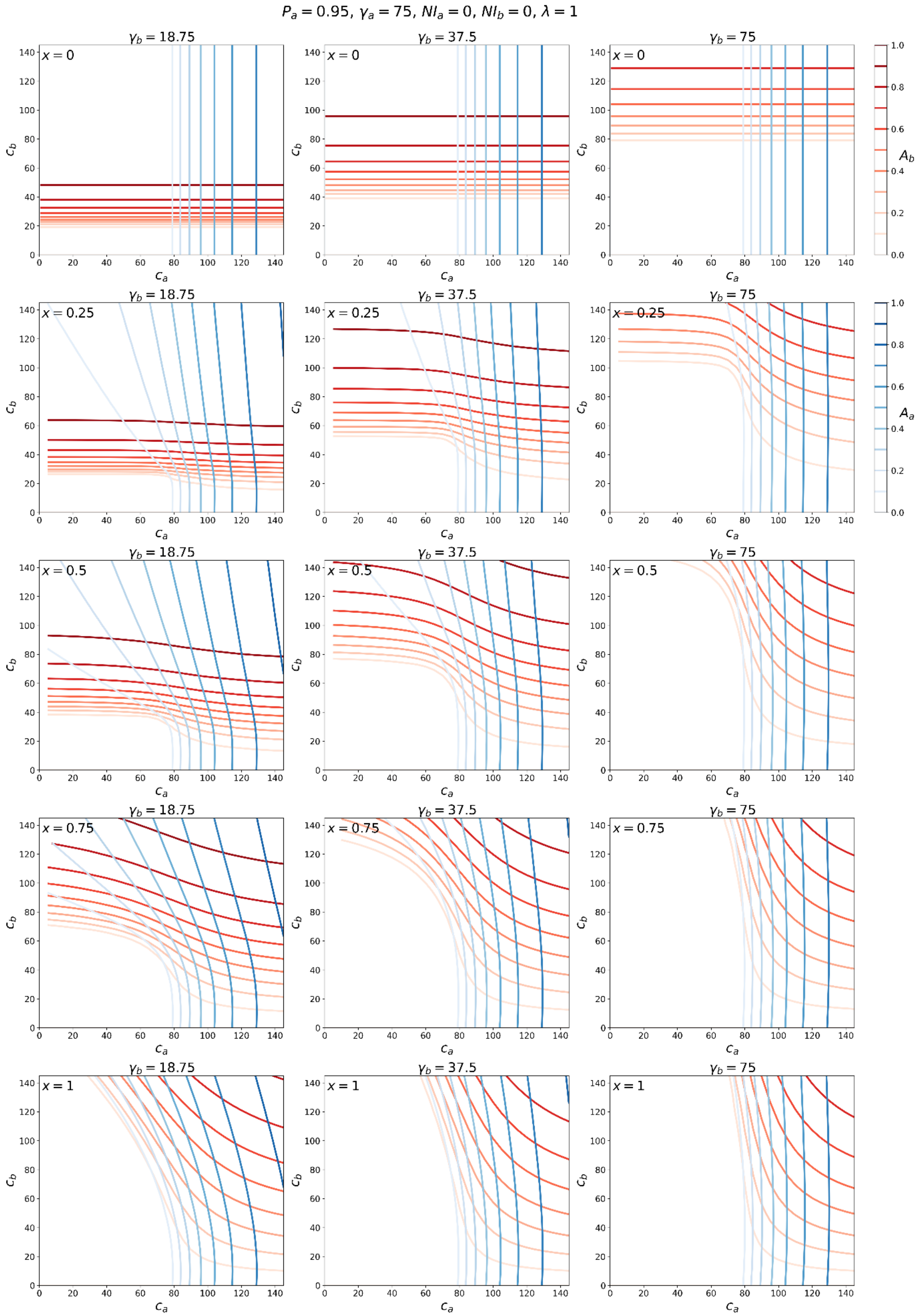


Figure 2: Contour maps of A_a (blue lines, see scale at the upper right) and A_b (red lines, see scale at the upper right) for a range of contact frequencies c_a and c_b. Each column of panels corresponds to a different γ_b and each row to a different x, as indicated.

Figs. 3-5 show the variation in the attack rates A_a and A_b as functions of x, for various (c_a, c_b) coordinates. Each panel is for one pair of the (c_a, c_b) coordinates, with c_a increasing (in columns) from left to right, and c_b decreasing (in rows) from top to bottom. In this way, one can visualize the behaviours of the attack rates with x, on the (c_a, c_b) plane, across a range of c_a and c_b values sampled from the phase diagrams shown in Fig. 2.

As can be seen, when $\gamma_b = \gamma_a/4$ (Fig. 3), increasing x decreases A_b for all values of (c_a, c_b) shown in the figure. The decrease in A_b can be dramatic, including going from $A_b = 1$ for small values of x to $A_b = 0$ for large values of x. Increasing x generally increases A_a, and the increase in A_a is largest for values of $c_a \leq \gamma_a$ (such that no epidemic would occur in the a group if it were completely isolated from the b group) and for intermediate values of x. When $c_a > \gamma_a$, increasing x has a very small effect on A_a, because the a group has a much larger population than the b group; this is reflected in the small changes in the blue contour lines in Fig. 2 for $c_a > \gamma_a$ and for increasing x.

When $\gamma_b = \gamma_a/2$ (Fig. 4), increasing x generally decreases A_b, similar to the results in Fig. 3, and x has a smaller effect on A_a compared to the results in Fig. 3. The only parameter values for which A_b increases with x are in the extreme lower-right corner of Fig. 4, for which (c_a, c_b) = (100, 25) and (125, 25). For these two pairs of (c_a, c_b) values, $c_b < \gamma_b$, such that the contact frequency of b individuals is so low that an epidemic would not occur among the vulnerable if they were completely excluded from the majority group. Furthermore, for (c_a, c_b) = (100, 25) and (125, 25), c_a is much greater than c_b, which is unrealistic given our interpretation of c_{ij} implied by our simplifying assumption $\beta_{aa} = \beta_{bb} = \beta_{ab} = \beta_{ba} = 1$ (see the Model section). We note that a similar, small increase in A_b versus x also occurs in the case of $\gamma_b = 18.75$ when $c_b < \gamma_b$ and $c_a >> c_b$, as can be seen in the left column of panels in Fig. 2, e.g. when $c_b \approx 15$ and $c_a = 120$.

When $\gamma_b = \gamma_a$ (Fig. 5), x has little effect on A_a, due to the differences in population sizes of the a and b groups. Increasing x can decrease A_b significantly when $c_b >> c_a$ (panels in the upper-left corner of Fig. 5) and can increase A_b significantly when $c_a >> c_b$ (panels in the lower-right corner of Fig. 5). This asymmetry occurs because of the asymmetry in population sizes N_a and N_b, causing $c_{ab} << c_{ba}$ (when $\lambda = 1$) such that it is much less likely for any given a person to come into contact with a b person than vice-versa. Similarly, in the right column of panels in Fig. 2, increasing x has a large effect on the red (A_b) contour lines and essentially no effect on the blue (A_a) contour lines.

In summary, increasing x for fixed c_b and c_a decreases the attack rate in the vulnerable group across all realistic values of the contact frequencies, when b represents a minority vulnerable population (here making up 5% of the total population and having a recovery time twice or four times as long as for the robust majority). This means that the vulnerable population is harmed by isolation from the robust population and benefits from mixing with or dilution within the robust population, in terms of risk of infection during the course of the epidemic or pandemic.

In the Appendices, we show that the same results hold when varying P_a, λ, and the seeding magnitude and distribution.

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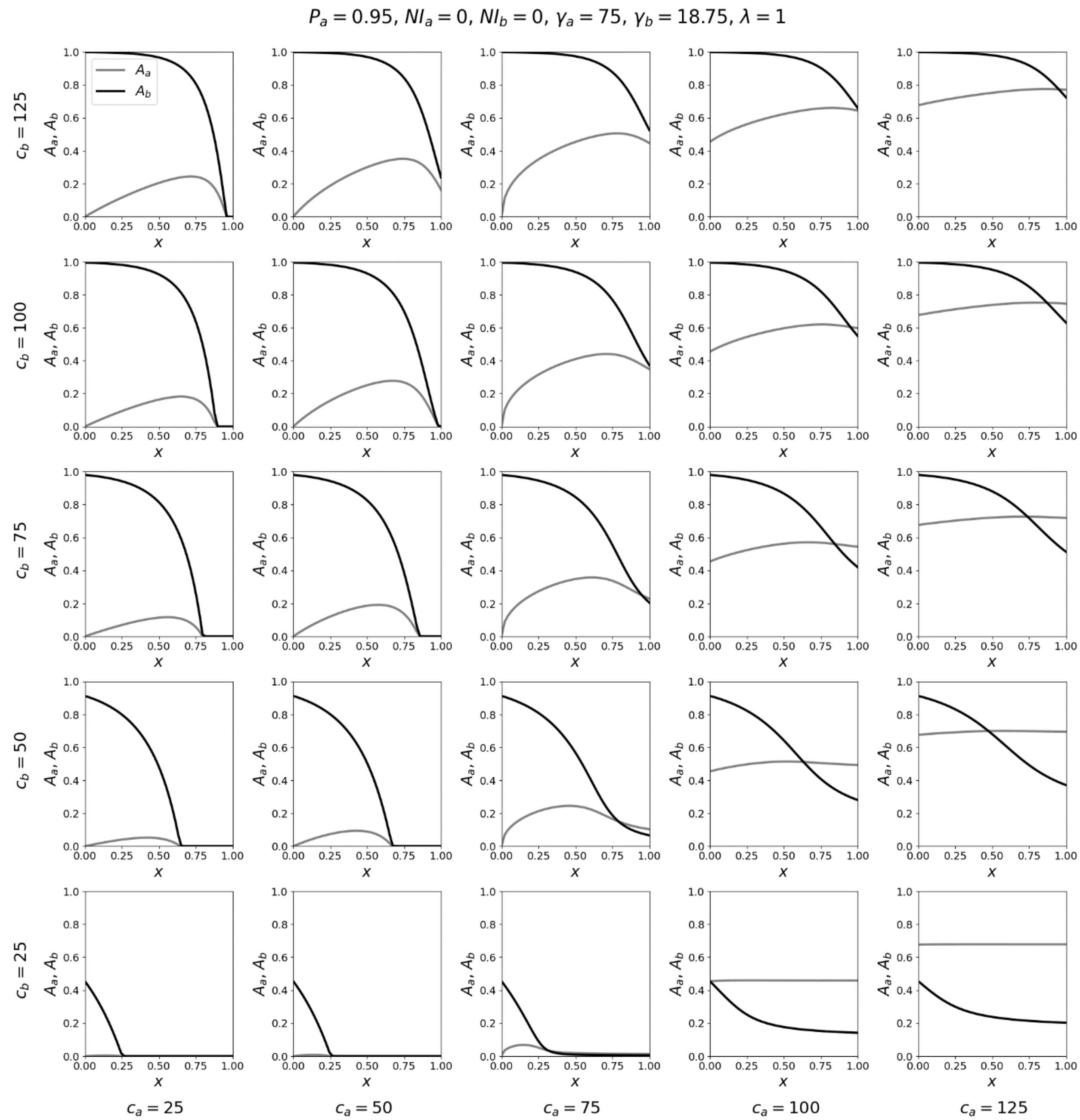


Figure 3: Attack-rates A_a and A_b as functions of x, for a range of contact frequencies c_a and c_b, for $\gamma_b = \gamma_a/4$.

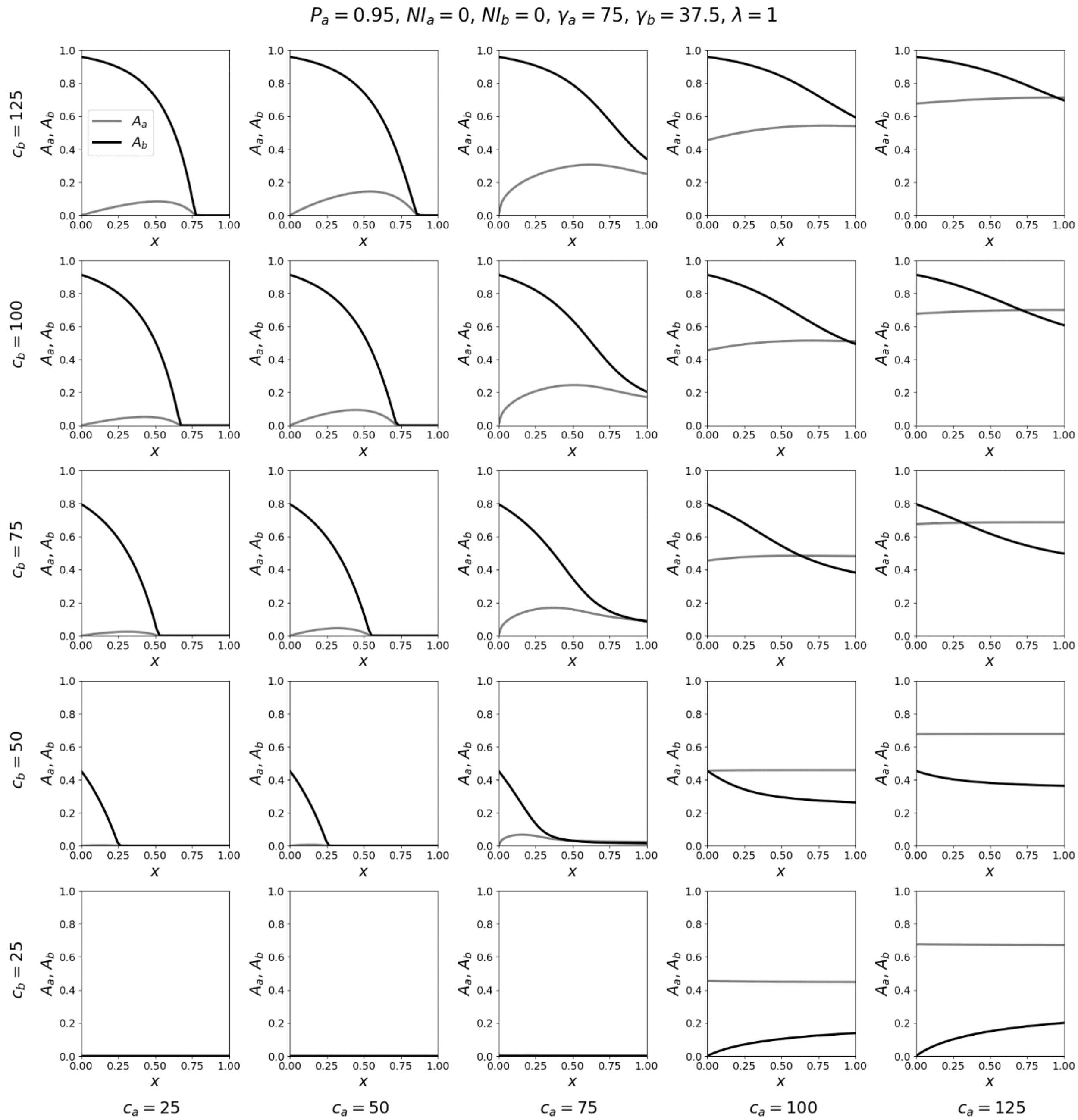


Figure 4: Same as Fig. 3, with $\gamma_b = \gamma_a/2$.

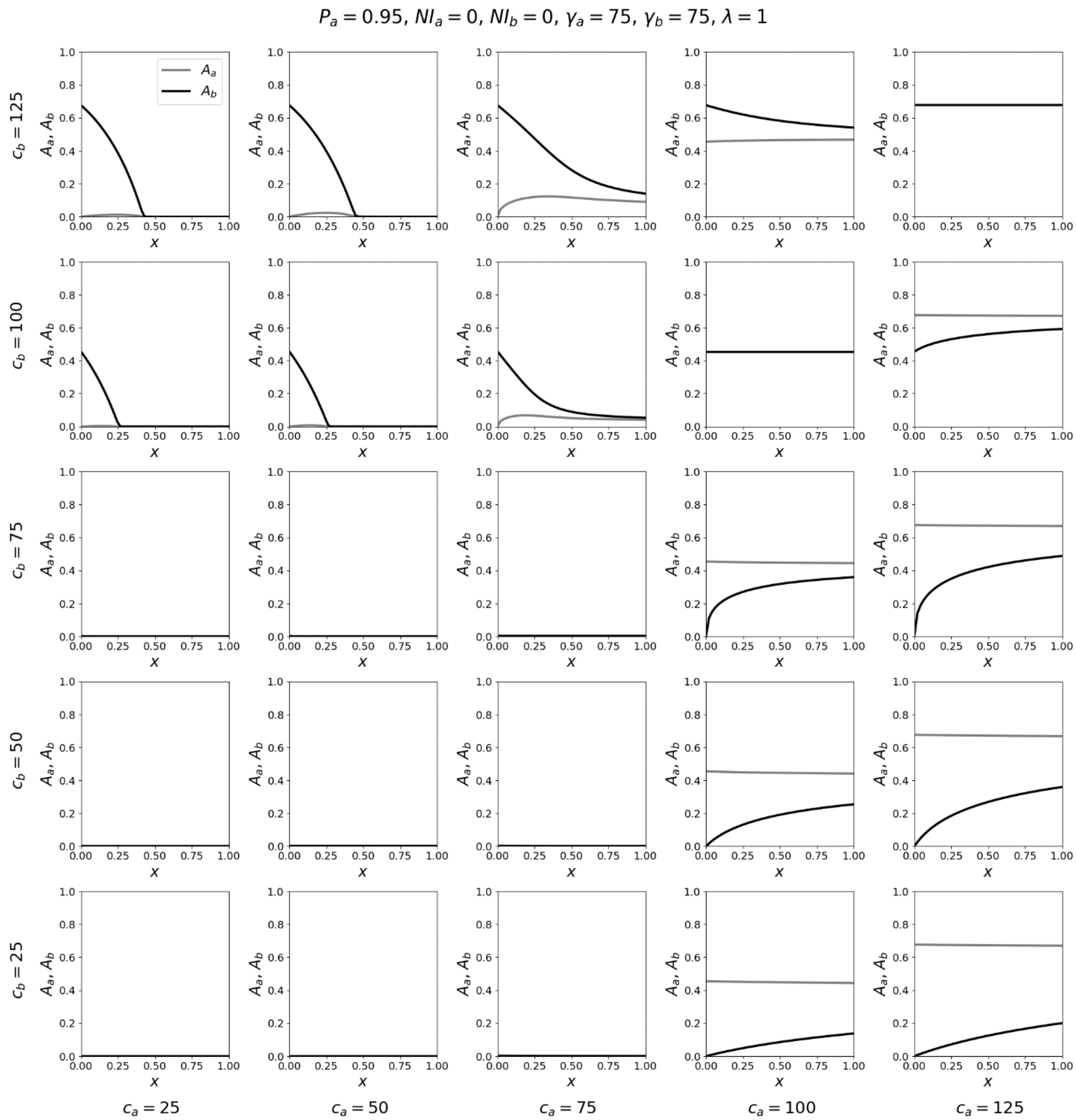


Figure 5: Same as Fig. 3, with $\gamma_b = \gamma_a$.

Discussion

Using a general two-population epidemic model, we have shown that increasing the degree of intermingling of the minority vulnerable (*b*) population with the majority robust (*a*) population reduces the attack-rate among the vulnerable. The advantage to the vulnerable group of intermingling with the robust group increases as the vulnerability of the minority group increases, that is, as their disease recovery time increases. Increasing the share of a vulnerable person's interactions that are with other vulnerable people, by confining them together in the same facility, increases the likelihood of infection of the vulnerable person during the course of the epidemic or pandemic, because infected vulnerable people remain infectious for a long time, relative to robust people.

The only exception to this general rule occurs if the contact frequency for vulnerable individuals is so small that no epidemic would occur in the vulnerable group if it were completely segregated from the robust majority of society, while the frequency of guaranteed infection-causing contacts for robust people is large enough to produce an epidemic in that group and is also much higher than that of vulnerable individuals. We expect this exception to be irrelevant in reality because it is unrealistic for $c_a >> c_b$, given the definition of the contact frequencies c_{ij} as representing contacts of sufficient physical proximity and duration such that a susceptible *i* person is guaranteed to be infected by an infectious *j* person (see the Model section).

Our analysis focuses on the two dominant and most fundamental features present in all epidemic models: the contact frequencies and recovery rates. On this simplest-possible yet sufficiently realistic foundation, we establish that segregating the vulnerable into care homes virtually always produces negative results in epidemic models. Not surprisingly, therefore, researchers using complex agent-based models have found that segregation of vulnerable individuals produces worse outcomes both for that group and for the society overall (Markovič et al., 2021).

Others have used simple epidemiological models to study segregation of "high-transmission-risk" and "low-transmission-risk" groups (Munday et al., 2018; Yuan et al., 2022; Garnett & Anderson, 1996). However, because such studies are focused on different transmission rates due to different behavioural and contact characteristics of the two groups – such as sexual preferences, cultural lifestyle factors, and willingness to become vaccinated – they do not consider the impact of different recovery rates for the two populations, which is crucial in the context of segregation of vulnerable individuals from the robust majority. Those studies, therefore, do not directly address the problem of society's vulnerable sector regarding infectious diseases.

Segregation based on vaccination status has also been studied recently using simple models (Hickey & Rancourt, 2022; Fisman et al., 2022; Virk, 2022; Kosinski, 2021). In this application, Hickey and Rancourt found that the effect of the segregation on increasing or decreasing the contact frequencies in the segregated groups is crucial and can cause the predicted epidemic outcomes to be worse for both the vaccinated and unvaccinated, compared to no segregation (Hickey & Rancourt, 2022). This highlights the importance of contact frequencies, which are necessarily impacted by segregation policies, and which again play a pivotal role in the present analysis.

Isolation policies intending to protect the vulnerable reduce their contacts with the outside world, for example by barring visitors from entering care homes and by reducing the frequency of interaction between care home staff and residents. The care home isolation policies are also designed to reduce the

number of epidemiological contacts between the care home residents themselves. However, since transmission of respiratory diseases is air-borne via long-lived suspended aerosol particles (Shaman & Kohn, 2009; Shaman et al., 2010) and occurs in indoor environments (Bulfone et al., 2021), confining many vulnerable people in the same facility in-effect increases the per-individual frequency of infectious contacts, because they are breathing the same air and ventilation is imperfect. Indeed, virtually all studied outbreaks of viral respiratory illnesses have occurred in indoor environments (Moser et al., 1979; Loeb et al., 2000; Salgado et al., 2002; Bulfone et al., 2021; Javid et al., 2021) and care homes for the elderly are known to be "ideal environments" for outbreaks of infectious respiratory diseases, due to the susceptibility of the residents living in close quarters (Strausbaugh et al., 2003; Gozalo et al., 2012; Lansbury et al., 2017). A policy that decrease c_{ba}, for example by barring younger family members from entering care homes to visit their elderly relatives, causes the isolated vulnerable people to spend more time in the care home, breathing the same air as the other residents. This in-effect increases c_{bb}.

For constant c_a, decreasing c_b reduces the attack rate in the vulnerable group, regardless of the value of x, as can be seen from Fig. 2. However, the sought decreasing of c_b is imposed by isolating the vulnerable (from society, loved ones and each other), which has important negative health consequences (Cohen et al., 1991; Cohen et al., 1997; Cohen, 2004; Holt-Lunstad et al., 2010; Holt-Lunstad et al., 2015; Valtorta et al., 2016). Psychosocial factors, including depression, lack of social support, and loneliness are known to play key roles in the negative health effects of isolation (Hemingway & Marmot, 1999; K.A. Matthews et al., 2010; Elovainio et al., 2017; Groarke et al., 2020; Spring et al., 2020). Proposed psychosocial factors uncovered by participatory qualitative research include dissonance between expectations and reality (Wang et al., 2020; Tarlov, 1996), which could be significant for vulnerable elderly patients with no prior life experience relevant to the isolation measures applied during the COVID era, which had no historical precedent.

Whereas governments used theoretical epidemic models to justify most public health policies during the COVID era, within a tunnel vision of reducing risk of infection with a particular virus, they appear not to have considered what those same models predict about infection rates under conditions of care home segregation; and they appear to have disregarded the exponential increase of infection fatality rate with age (COVID-19 Forecasting Team, 2022). Care home segregation policies may have been responsible for many deaths attributed to COVID-19 in Western countries.

We conclude that segregation and isolation of the vulnerable into care homes as a strategy to reduce the risk of infection during the course of an epidemic or pandemic is contrary to the most relevant immediate considerations from epidemiological models, in realistic conditions in which vulnerable people are highly susceptible and take longer to recover. The model parameter space, within possible parameter values, is one where it is virtually never epidemiologically advantageous to segregate and isolate frail people.

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These are the Appendices of the pre-print "Predictions from standard epidemiological models of consequences of segregating and isolating vulnerable people into care facilities" by J. Hickey & D.G. Rancourt, uploaded to https://www.medrxiv.org/ on 2023-02-05.

Appendices

Table of Contents

Table of Symbols

Symbol	Description
N_a	Total number of *a* individuals
N_b	Total number of *b* individuals
P_a	Proportion of the total population that belongs to the *a* group
λ	Coefficient modulating the relationship between c_{ab} and c_{ba}, as per Eq. 2 of the main text
γ_a	Recovery rate of infected *a* individuals
γ_b	Recovery rate of infected *b* individuals
seed	Total number of infected individuals at the outset of the simulation
ssa	Share of all seed individuals who belong to the *a* group
NI_a	Natural immunity of *a* individuals (set to 0 in all results shown in the main text and the appendices)
NI_b	Natural immunity of *b* individuals (set to 0 in all results shown in the main text and the appendices)
c_a	Frequency of contacts involving an *a* individual *
c_{aa}	Frequency of contacts between two *a* individuals *
c_{ab}	Frequency of contacts between an *a* and a *b* individual *
c_b	Frequency of contacts involving an *a* individual *
c_{bb}	Frequency of contacts between two *b* individuals *
c_{ba}	Frequency of contacts between an *b* and an *a* individual *
A_a	Attack rate among the *a* population (Eq. 3, main text)
A_b	Attack rate among the *b* population (Eq. 3, main text)
x	Degree of segregation versus intermingling of the *a* and *b* groups (Eq. 4, main text)

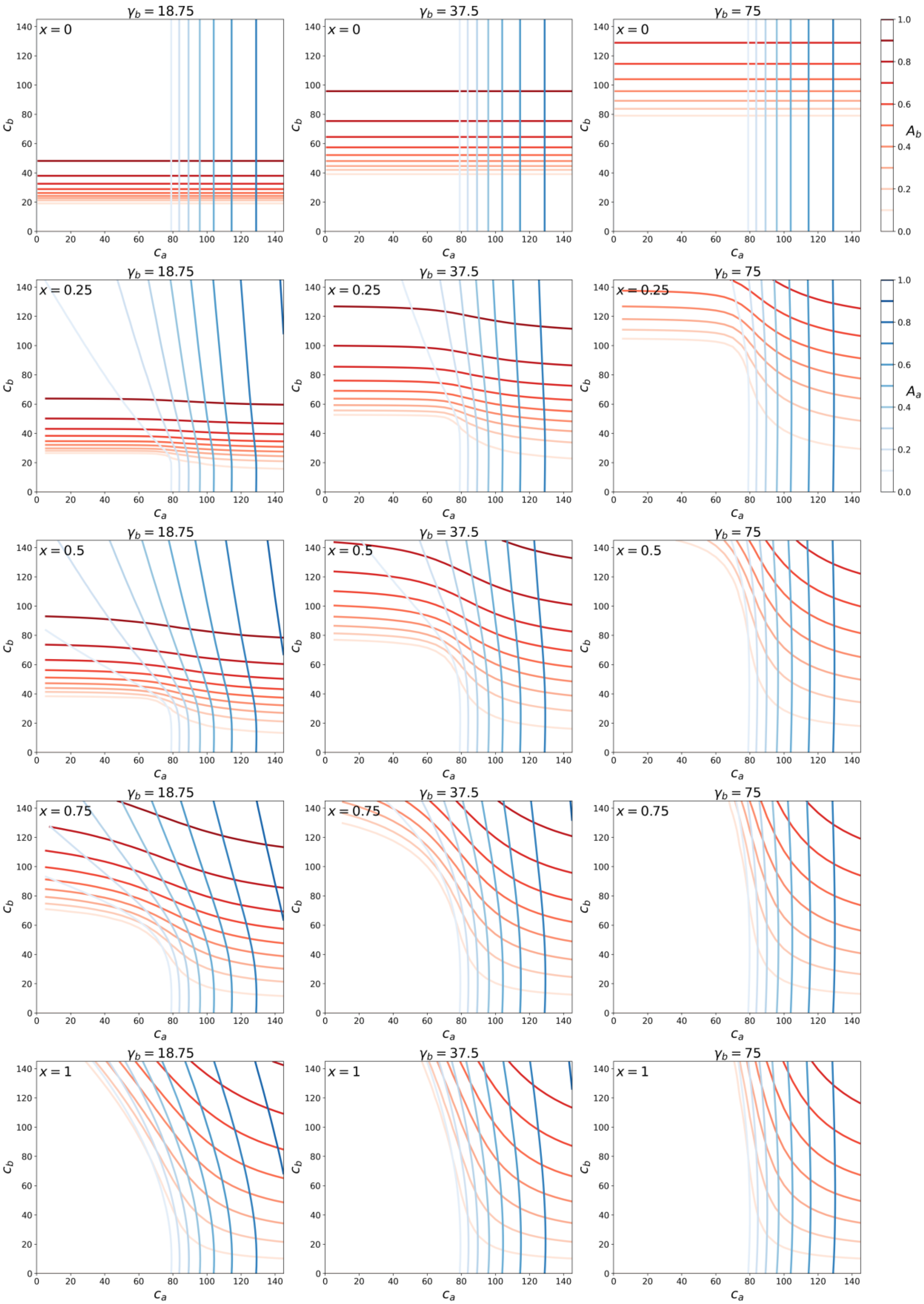
* Note that contact frequencies c_{ij} (i.e., c_{aa}, c_{ab}, c_{bb}, and c_{ba}) are defined such that the contact is guaranteed to result in infection when the contact is between a susceptible *i* person and an infectious *j* person, and that $c_a = c_{aa} + c_{ab}$ and $c_b = c_{bb} + c_{ba}$, as explained in the main text (Model section).

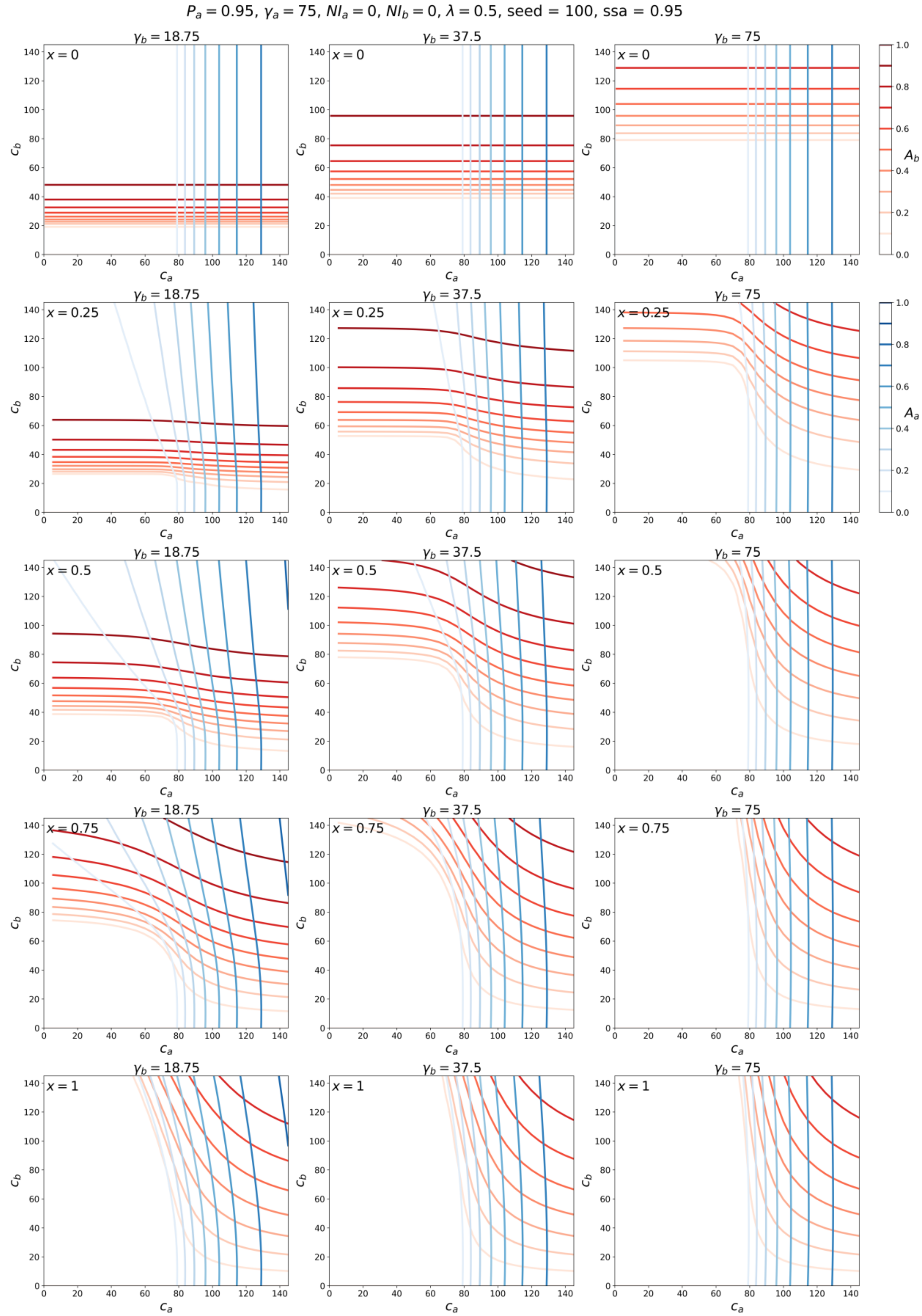
Appendix A: Additional results for P_a = 0.95

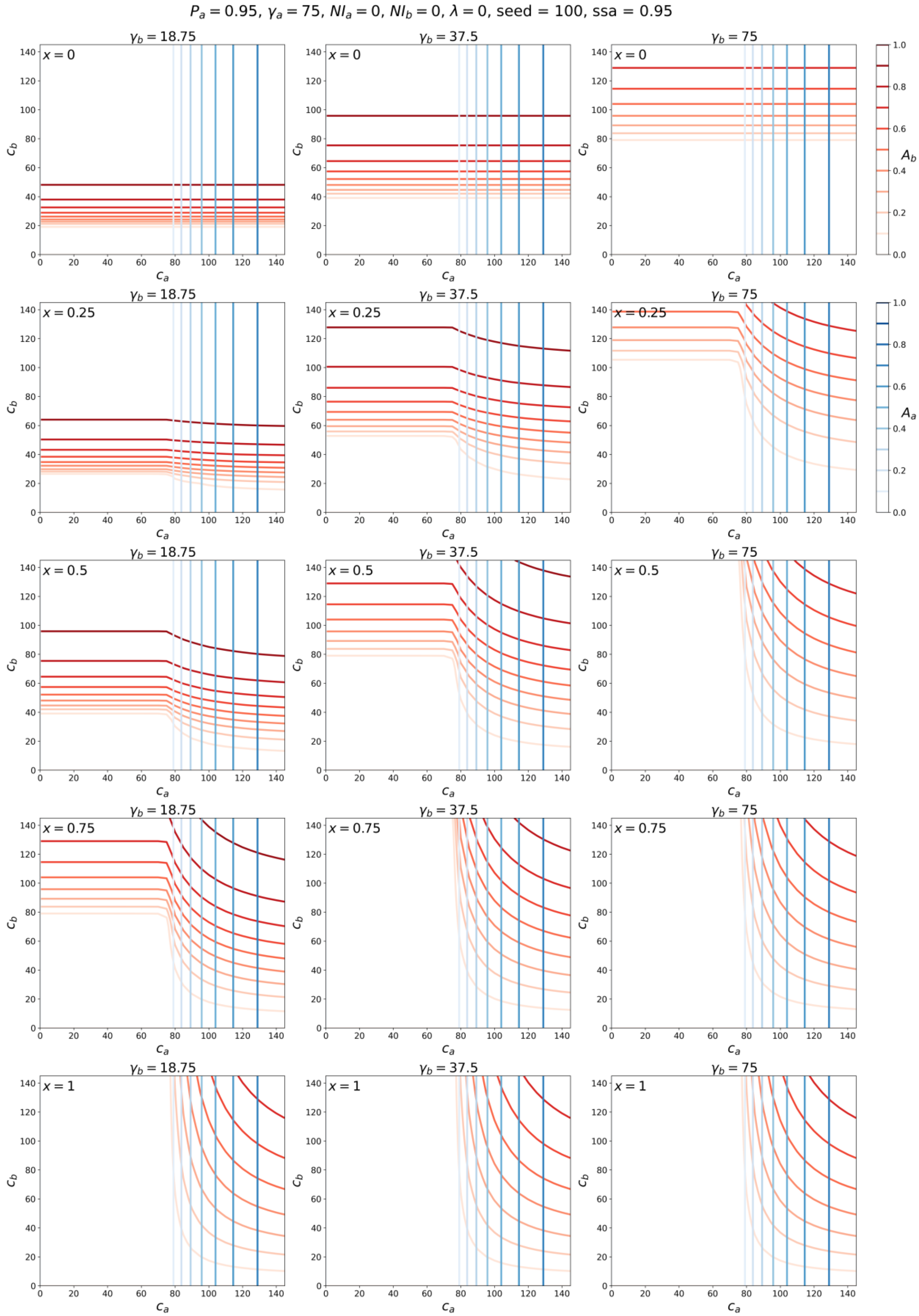
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A.1: Attack-rate contour maps for different values of λ

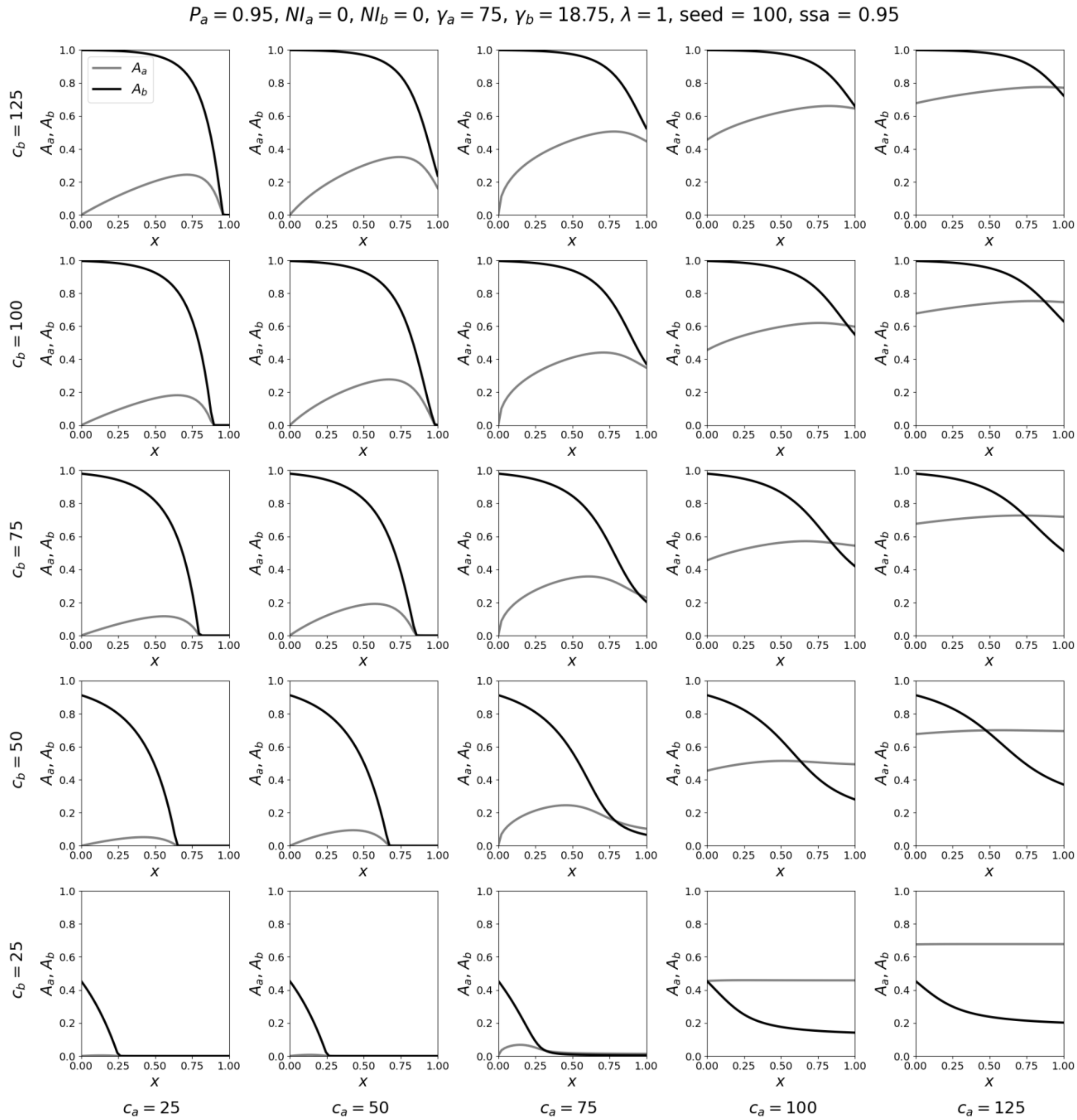
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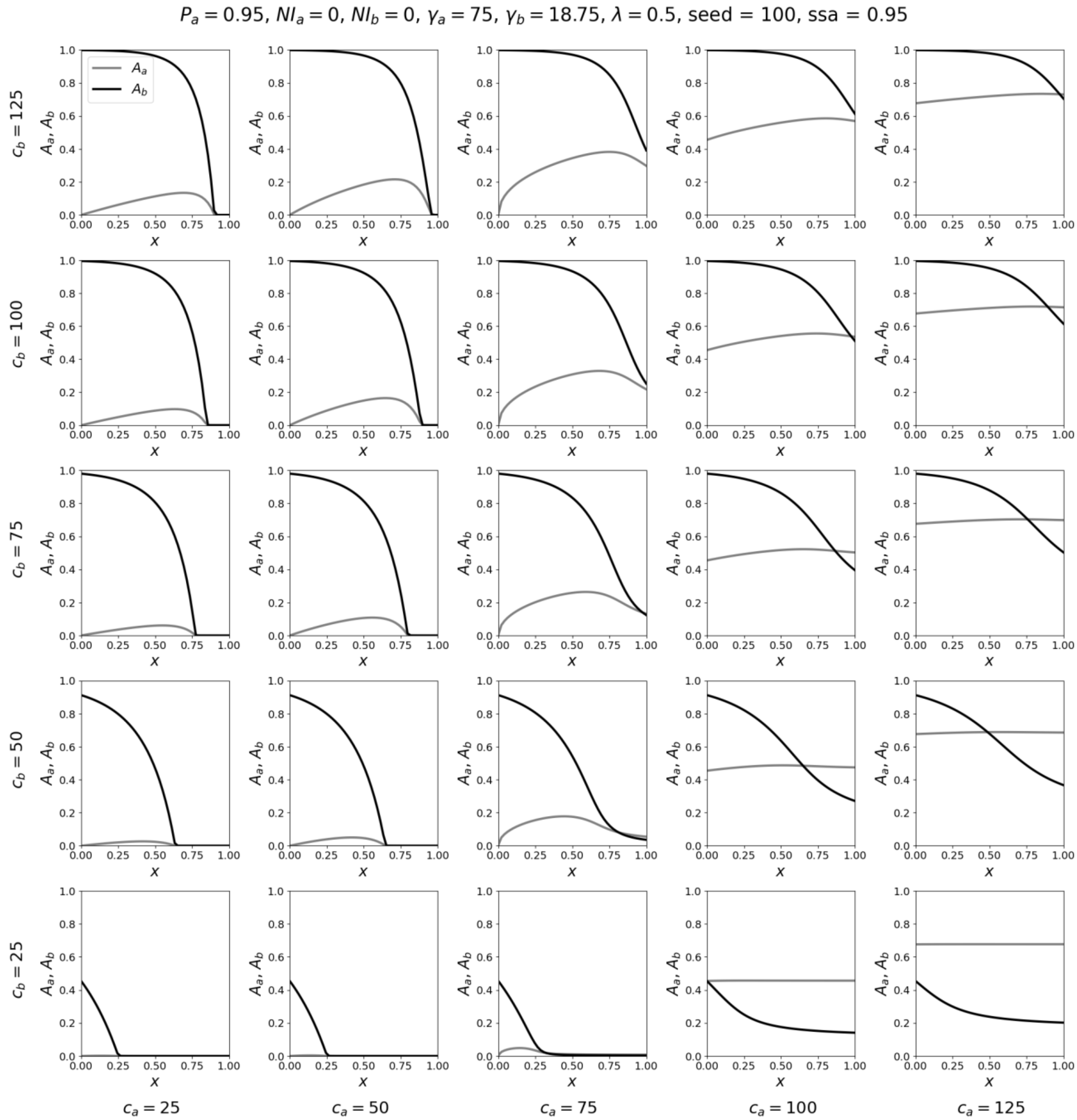


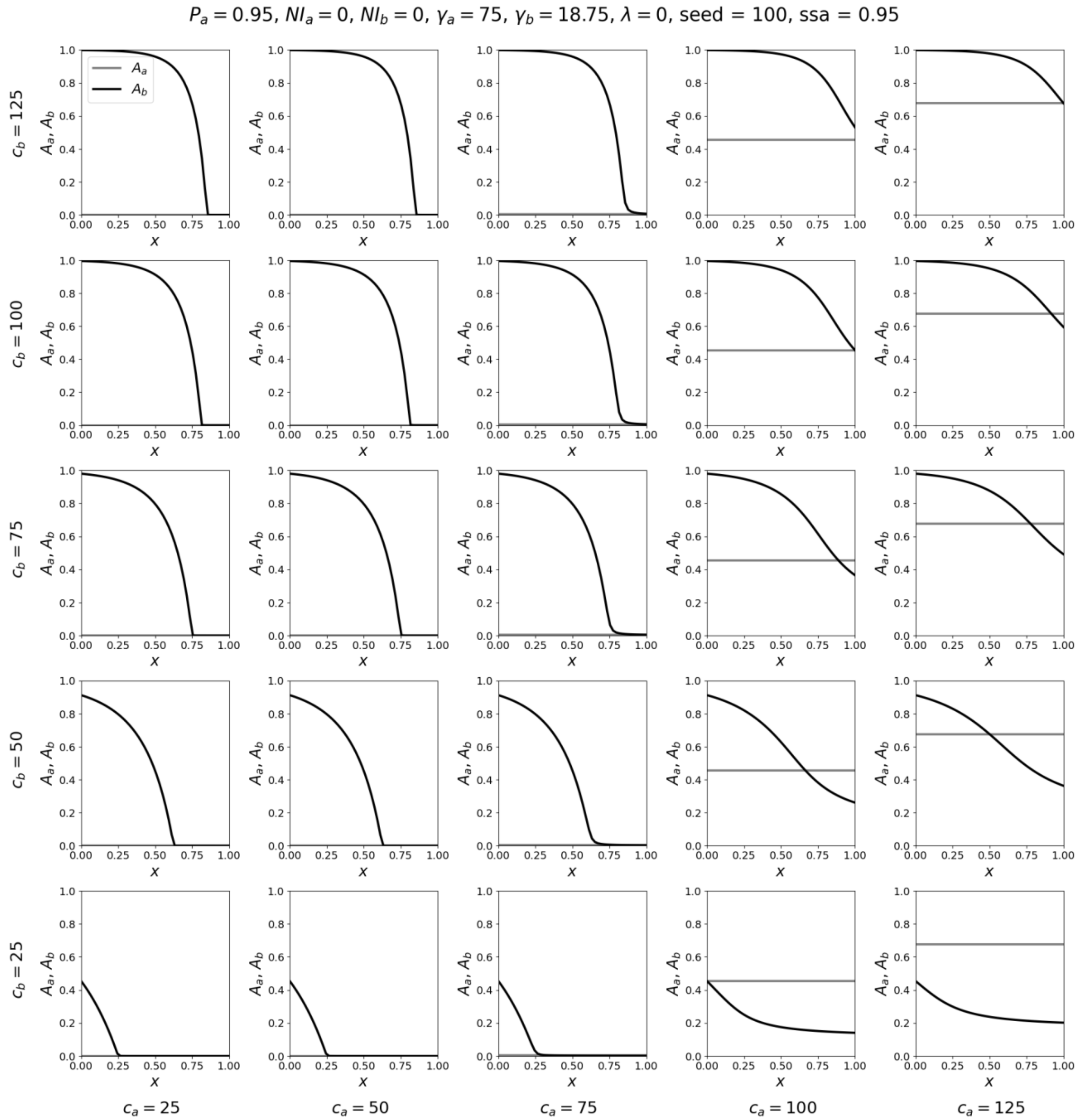

$P_a = 0.95$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0.5$, seed = 100, ssa = 0.95
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

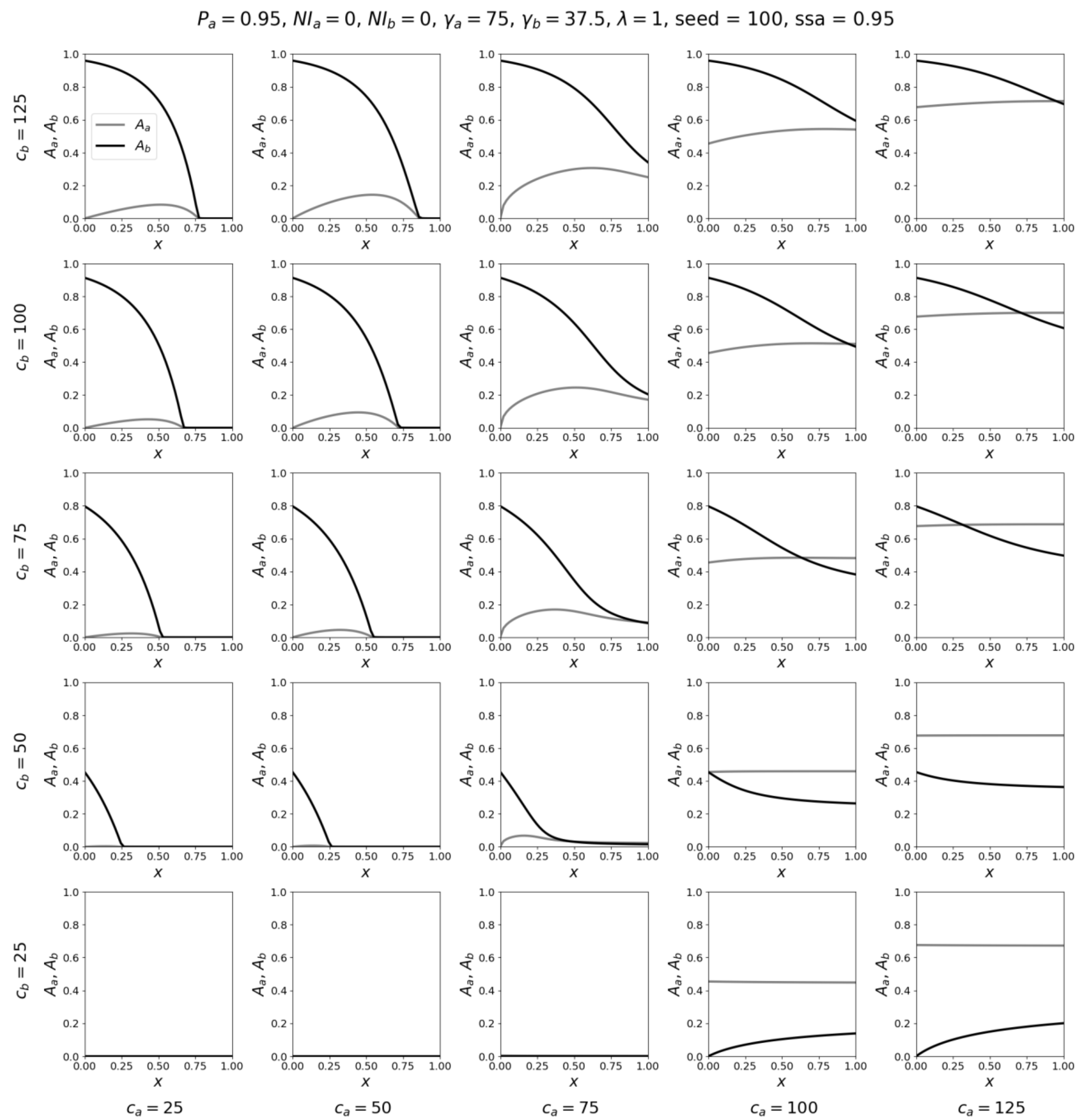

$P_a = 0.95$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0$, seed = 100, ssa = 0.95
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

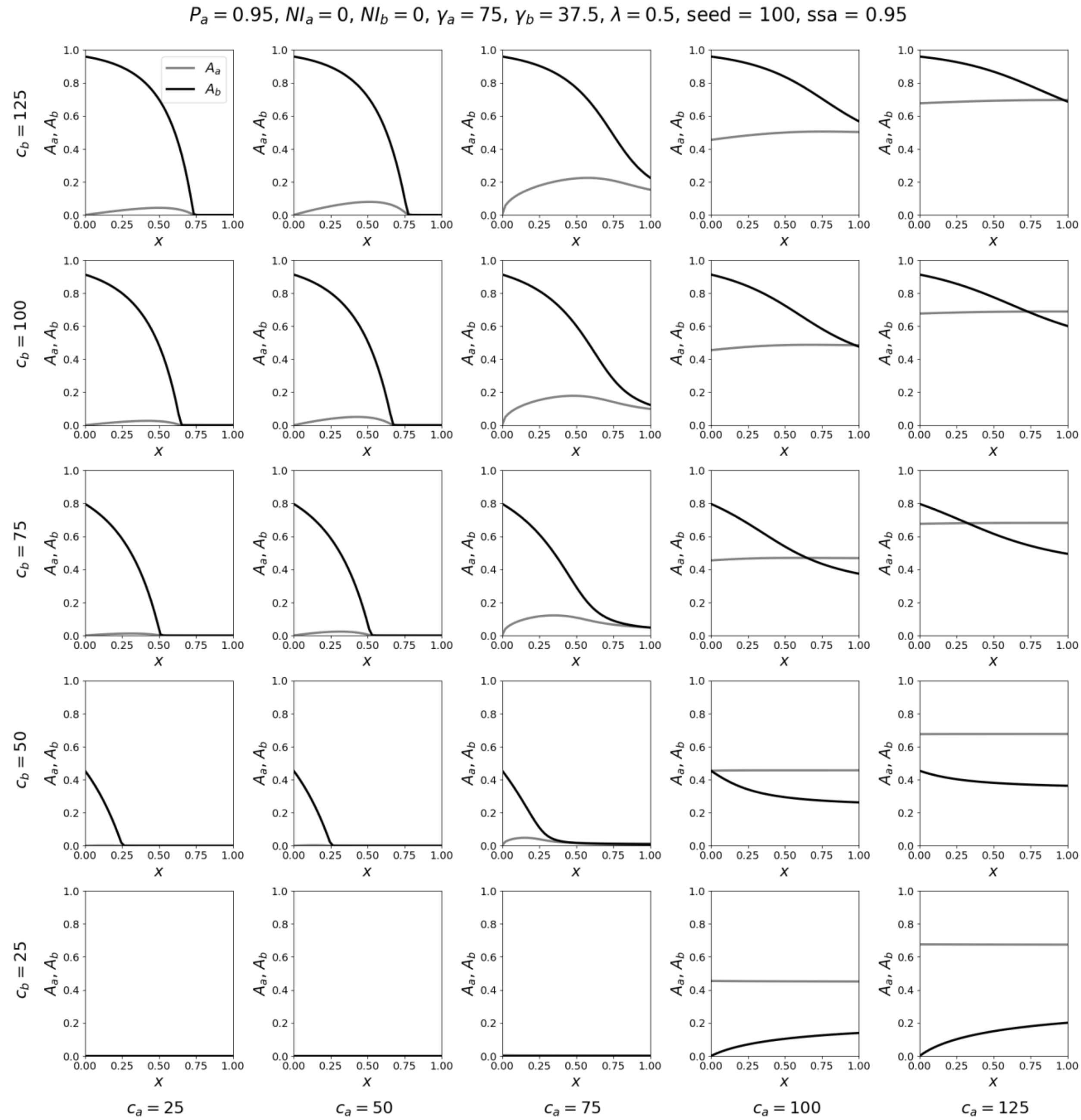
A.2: Attack-rate vs. x composite plots, for different values of λ

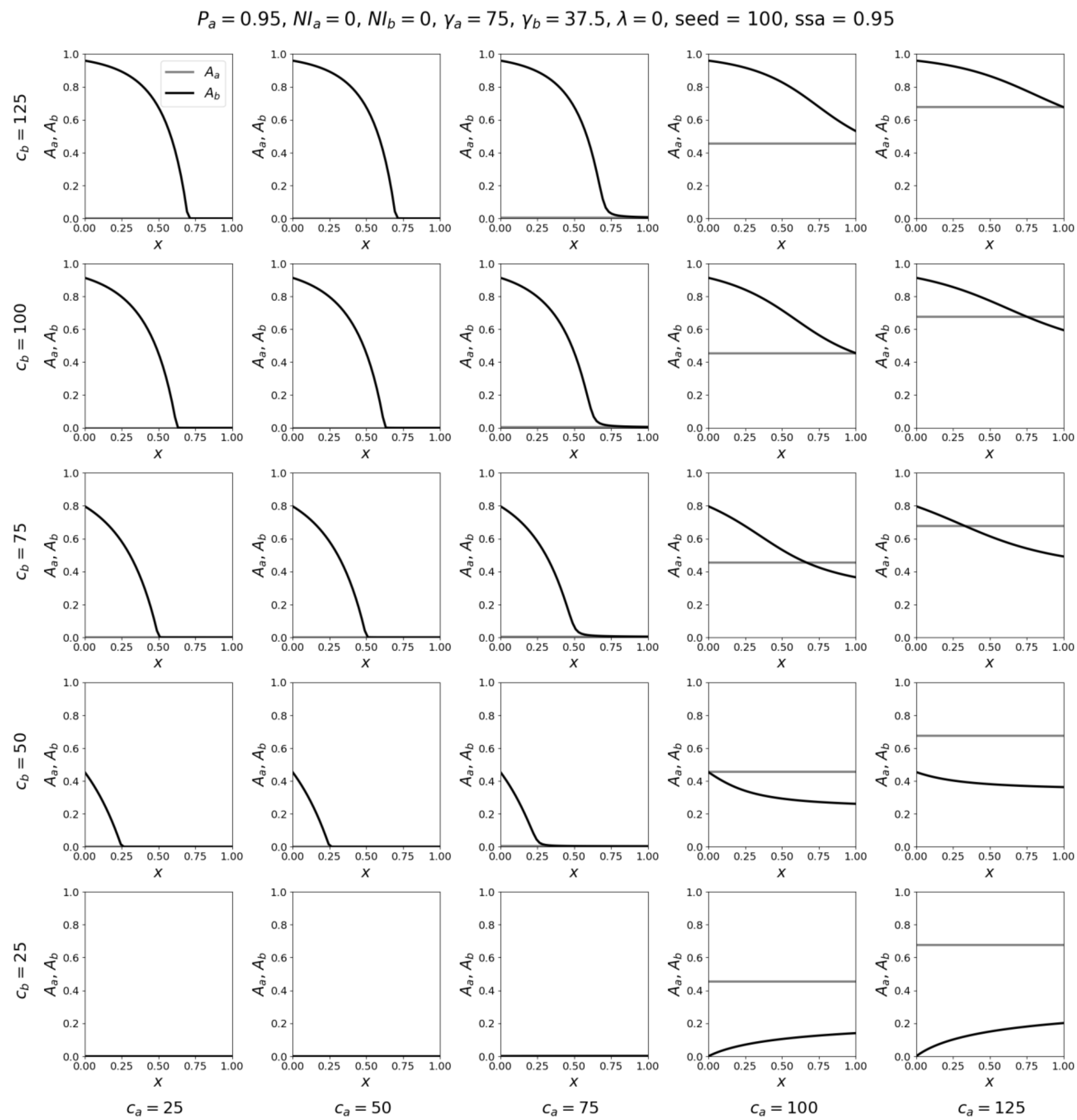

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A_a
A_b
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
A_a, A_b
x
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$

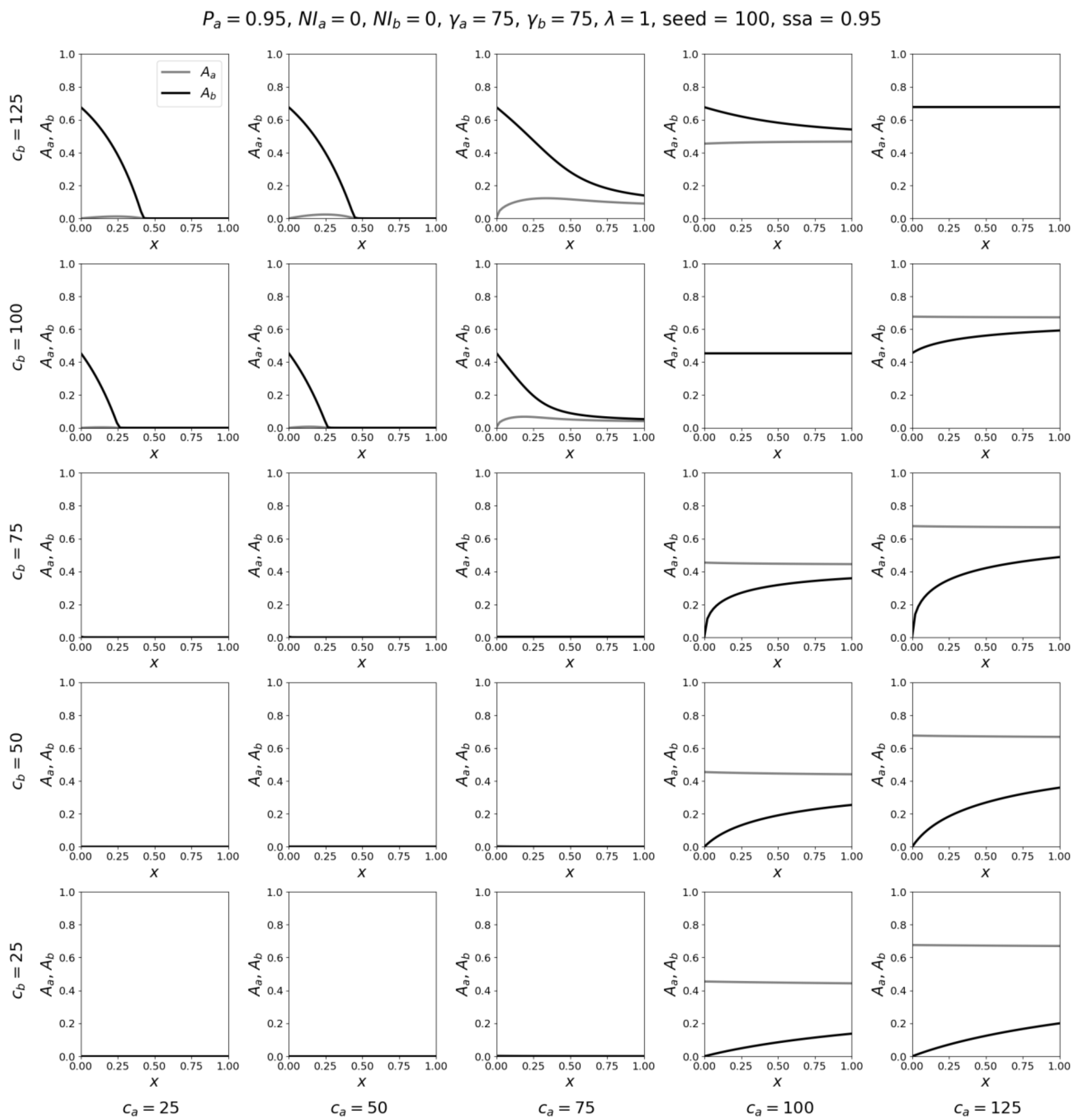

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A_a
A_b
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
A_a, A_b
x
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$


$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 18.75$, $\lambda = 0$, seed = 100, ssa = 0.95
A_a
A_b
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
A_a, A_b
x
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$

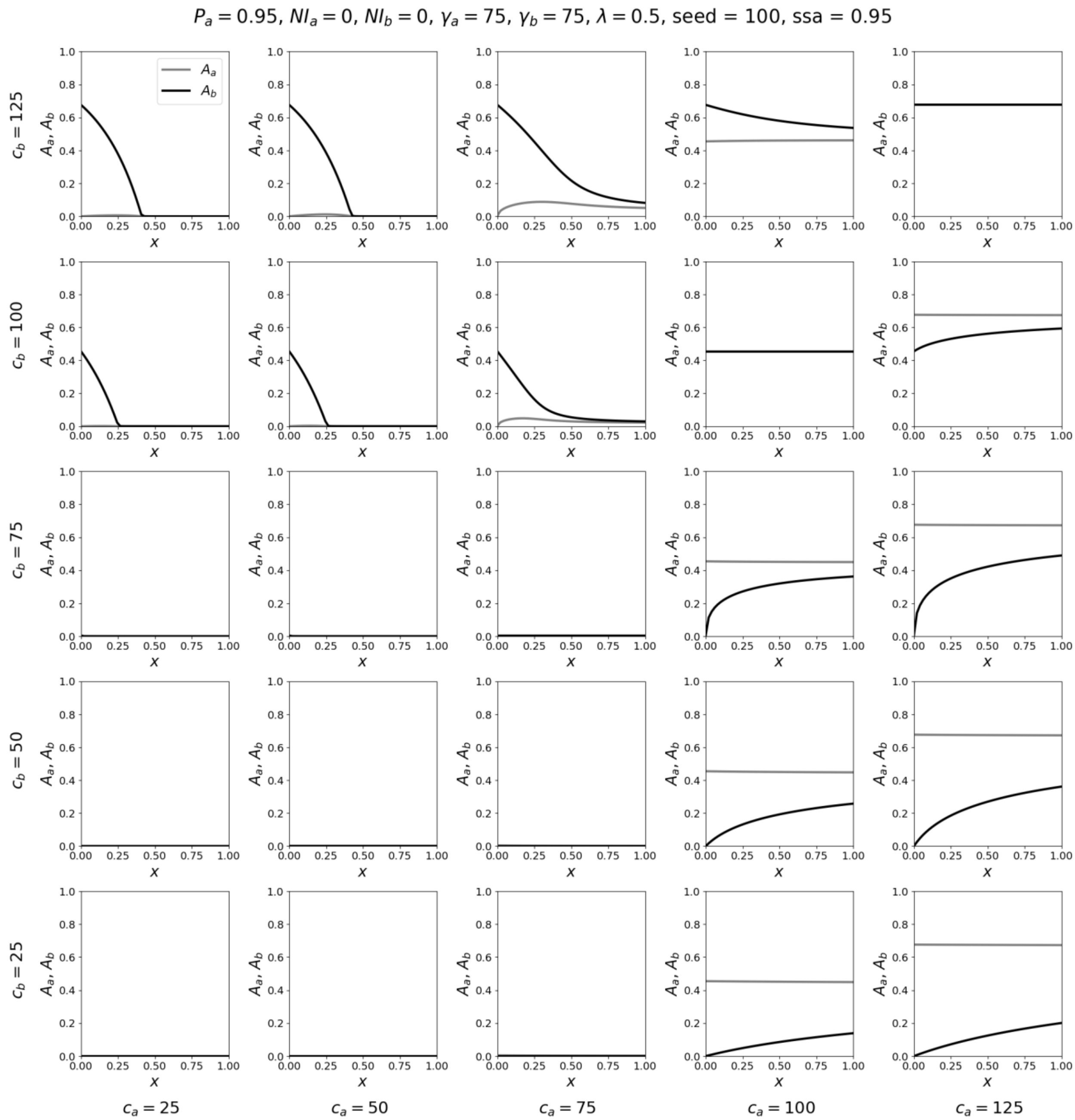

$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 37.5$, $\lambda = 1$, seed = 100, ssa = 0.95
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
A_a, A_b
A_a
A_b
x
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$

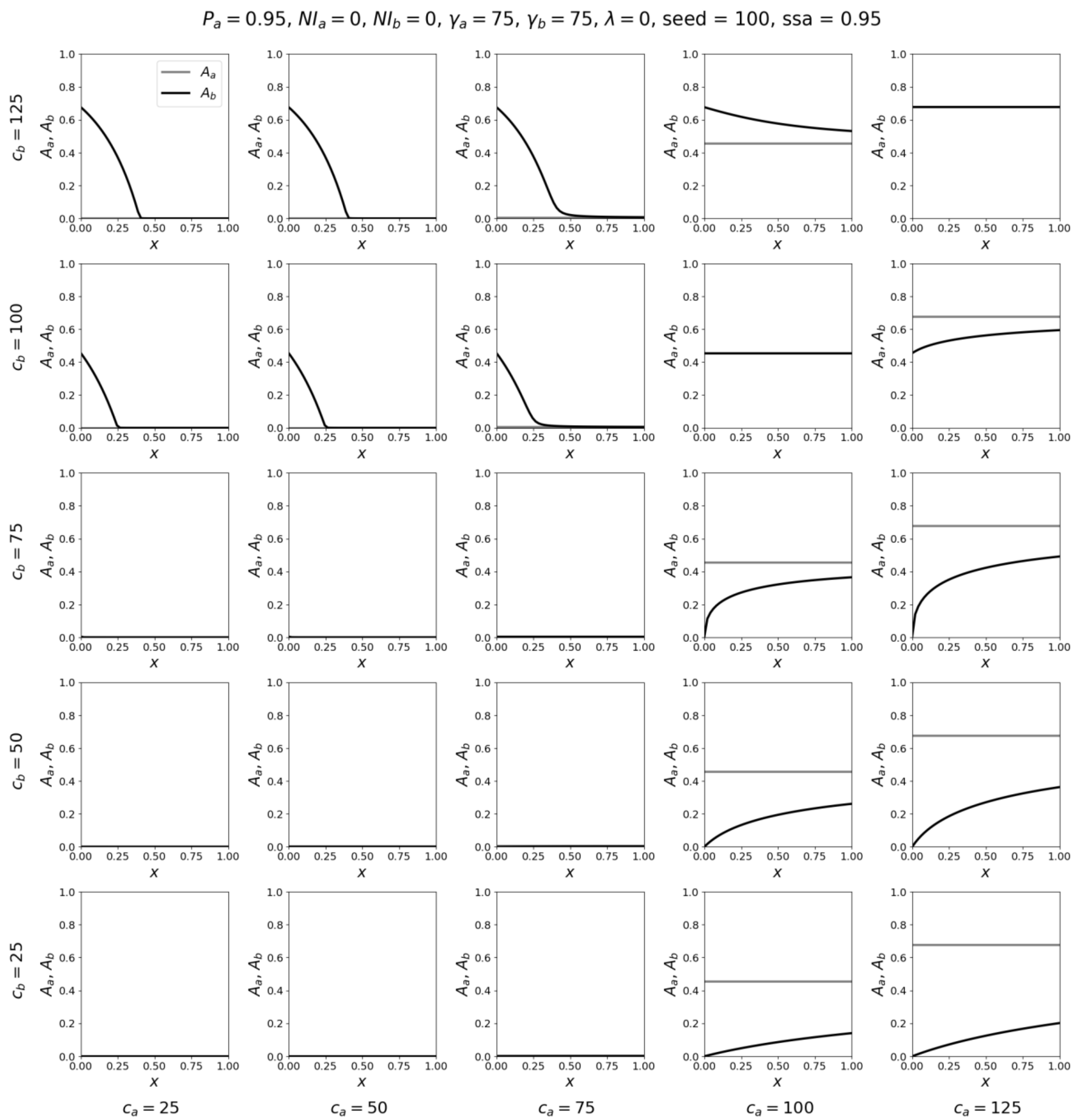

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A_a
A_b
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
A_a, A_b
x
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$


$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 37.5$, $\lambda = 0$, seed = 100, ssa = 0.95
A_a
A_b
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
A_a, A_b
x
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$

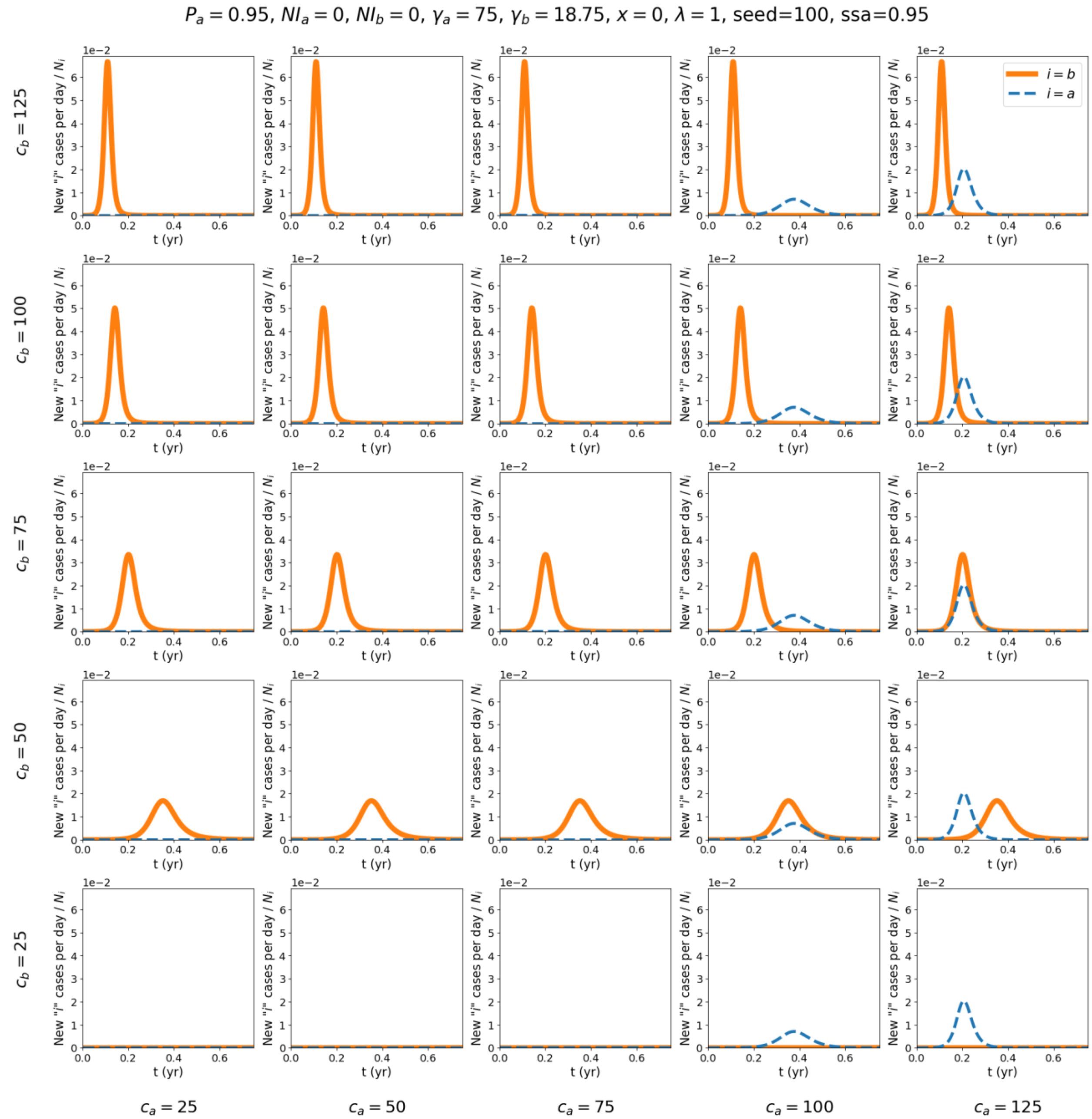

$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 75$, $\lambda = 1$, seed = 100, ssa = 0.95
A_a
A_b
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
A_a, A_b
x
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$

75

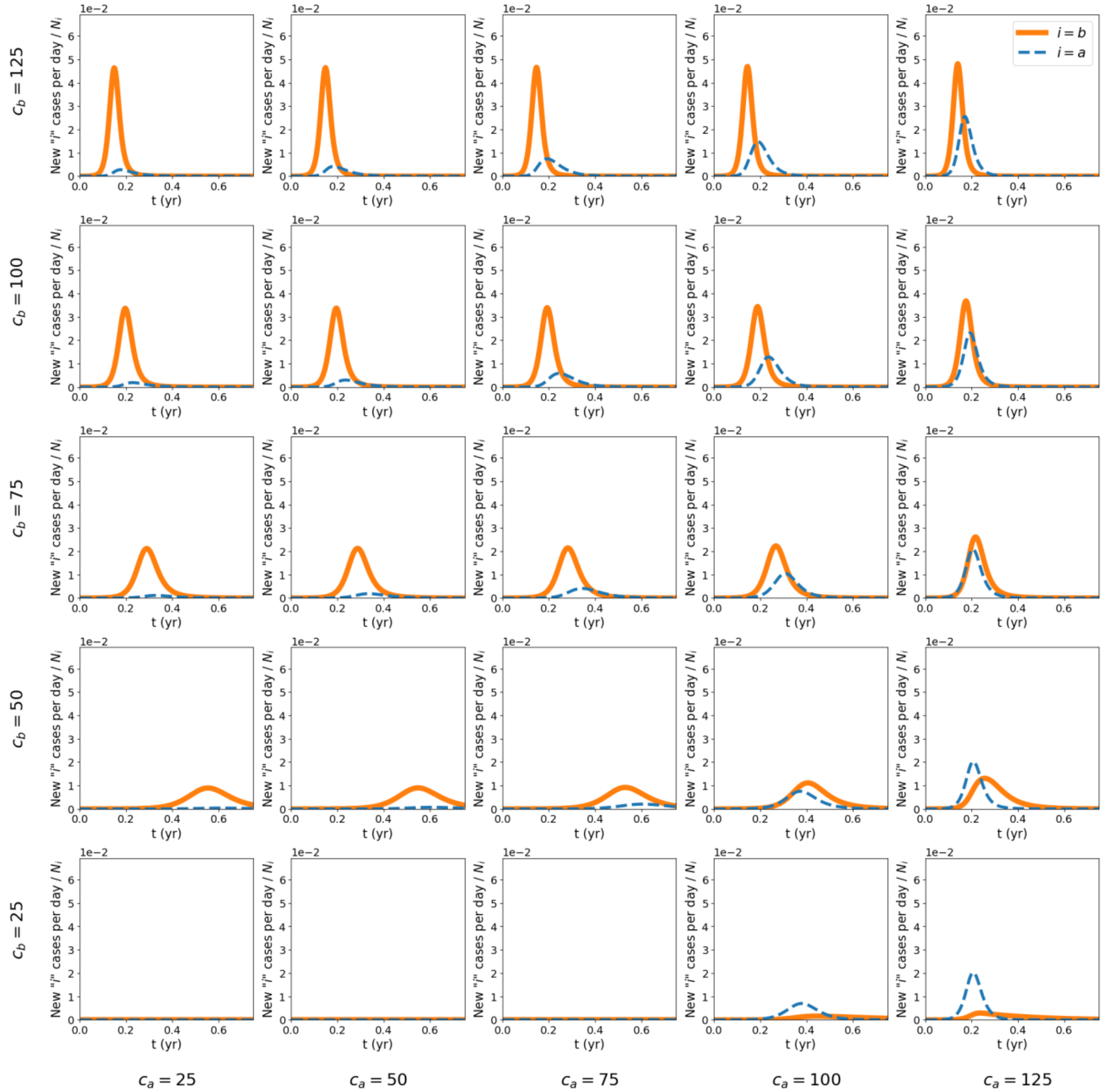

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A_a
A_b
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
A_a, A_b
x
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$

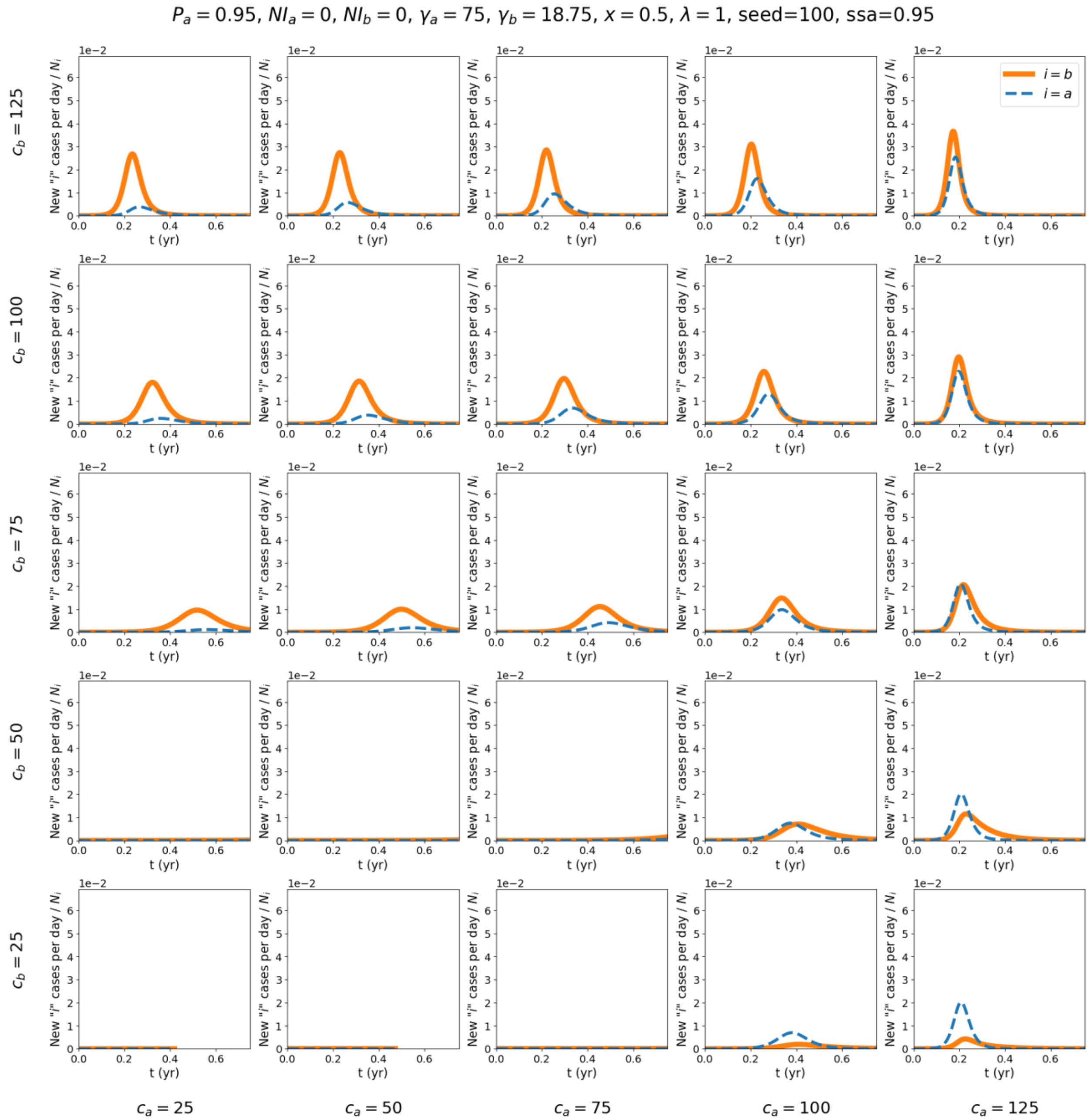

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A_a
A_b
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
A_a, A_b
x

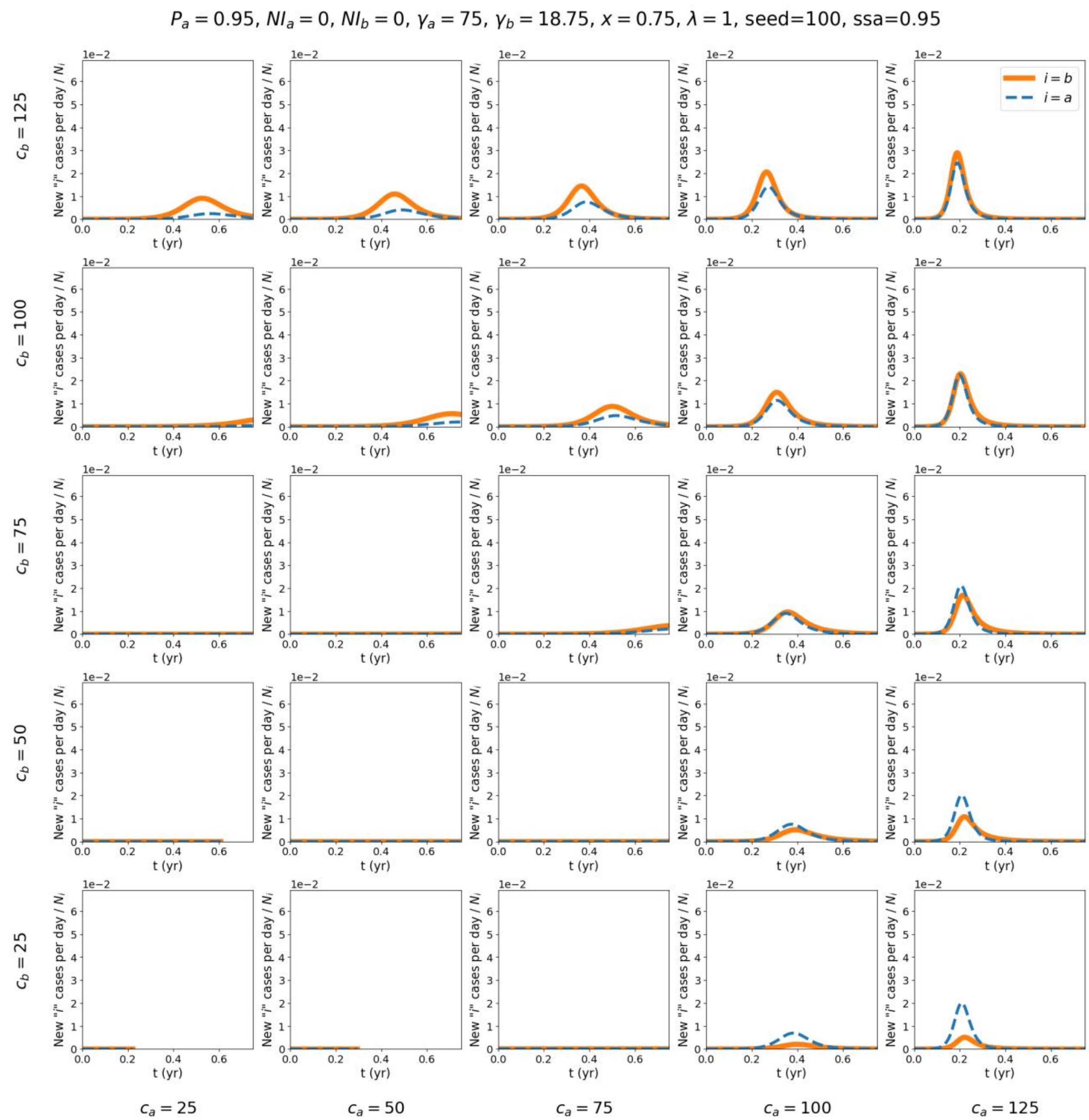
A.3: Epidemic curves for different values of x, for λ = 1

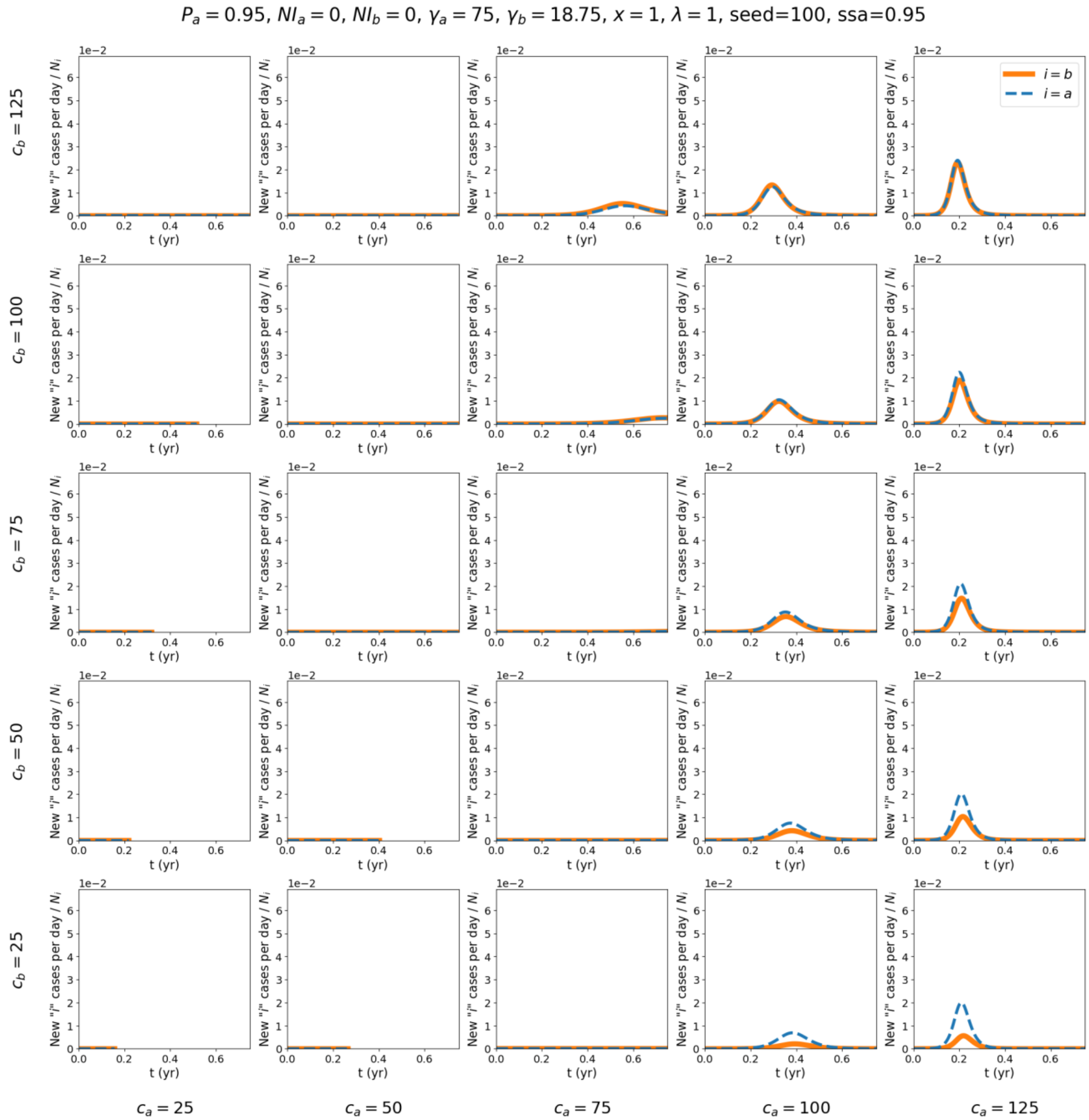

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$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
$i = b$
$i = a$
New "i" cases per day / N_i
t (yr)
1e−2

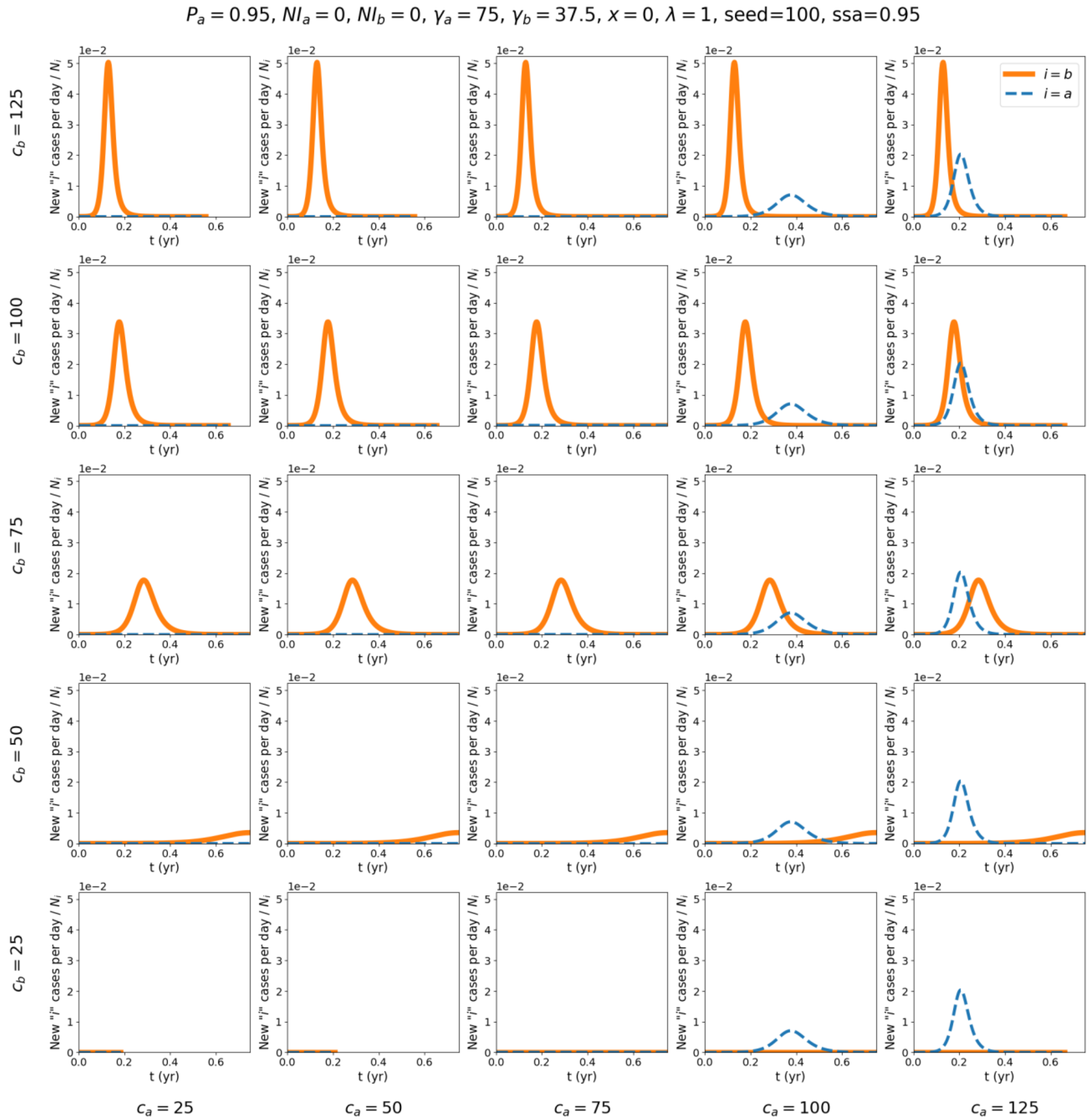
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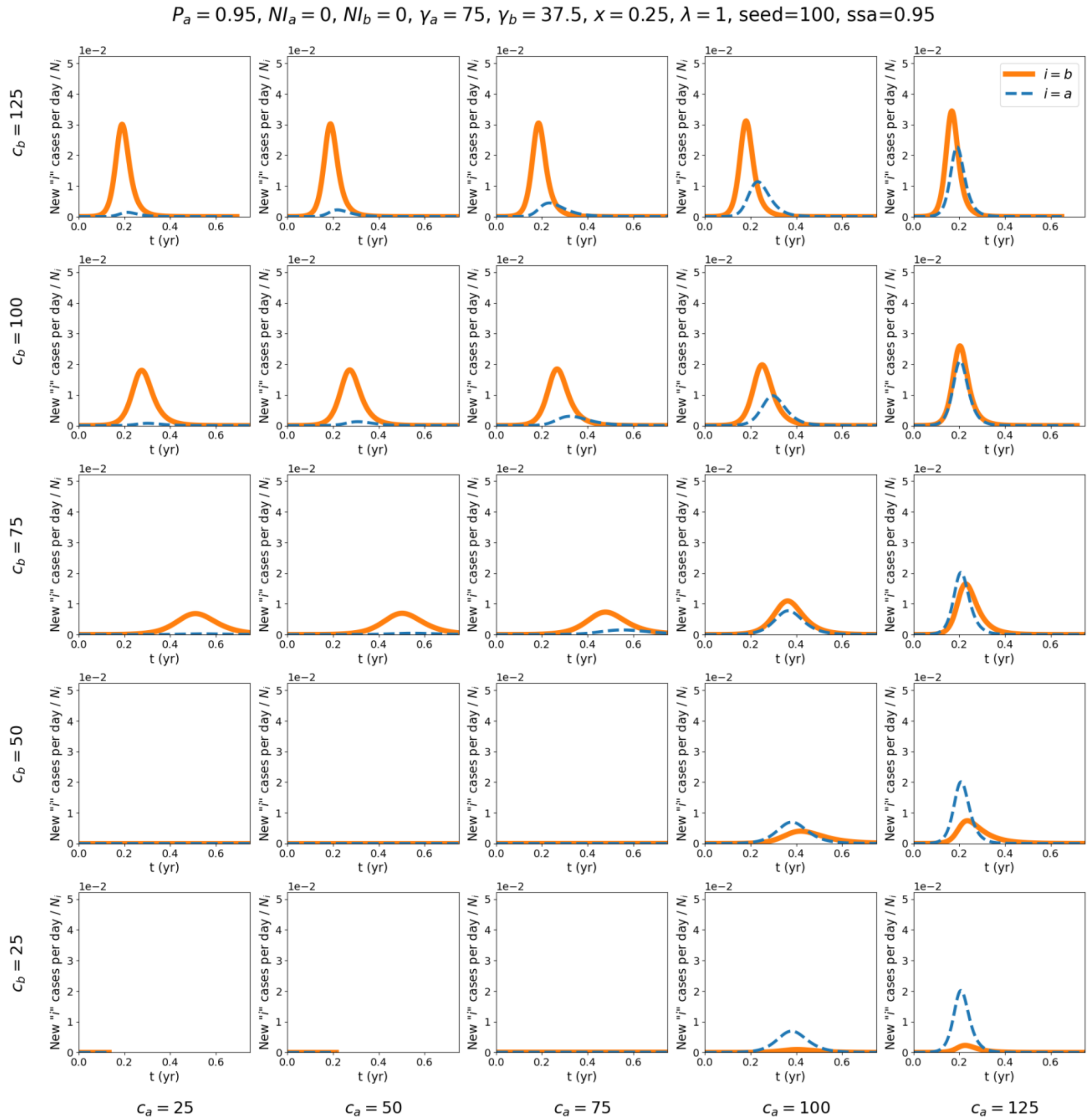


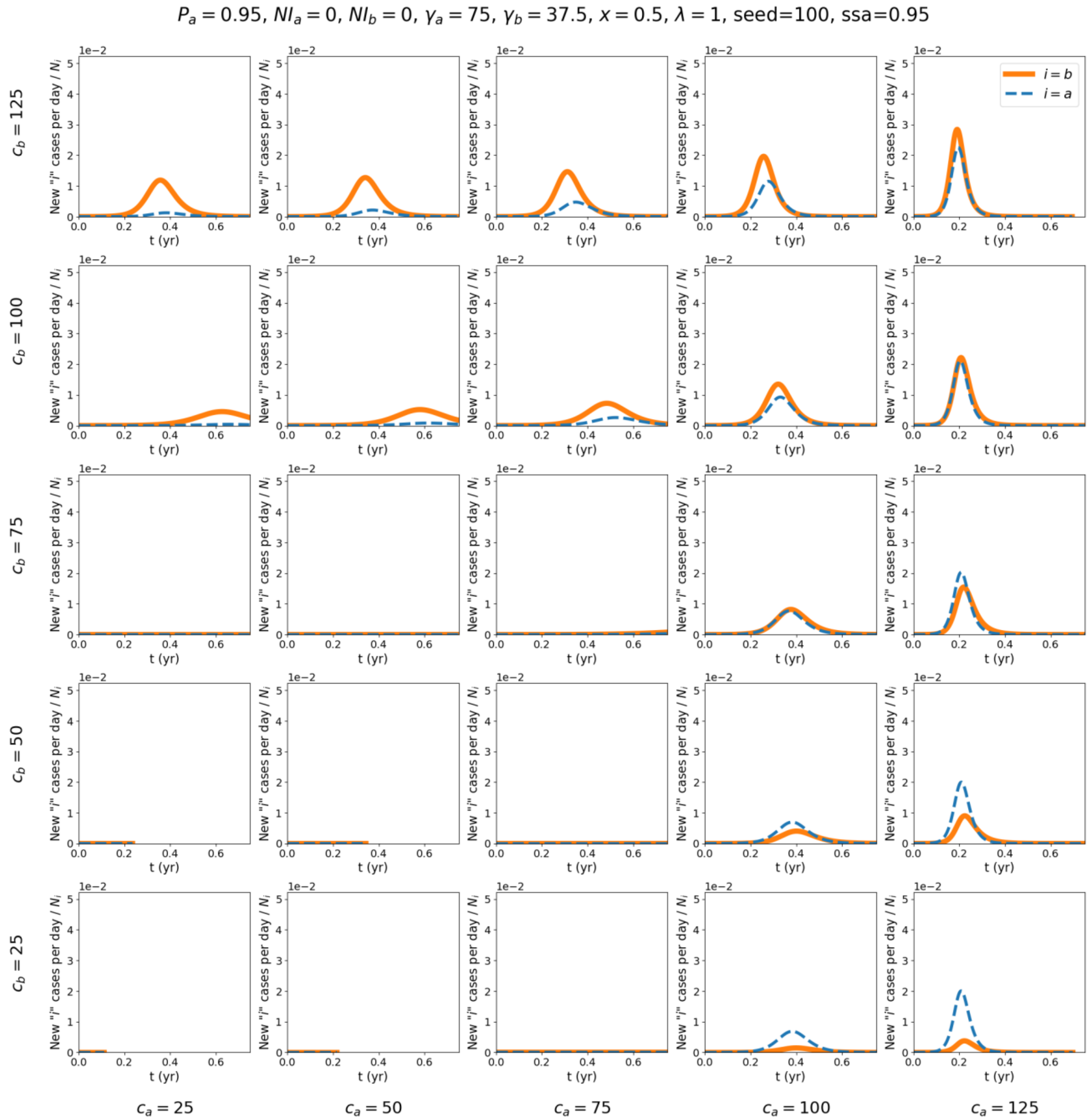

$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 18.75$, $x = 0.5$, $\lambda = 1$, seed=100, ssa=0.95
$i = b$
$i = a$
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
New "i" cases per day / N_i
t (yr)
1e−2
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$


$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 18.75$, $x = 0.75$, $\lambda = 1$, seed=100, ssa=0.95
$i = b$
$i = a$
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2

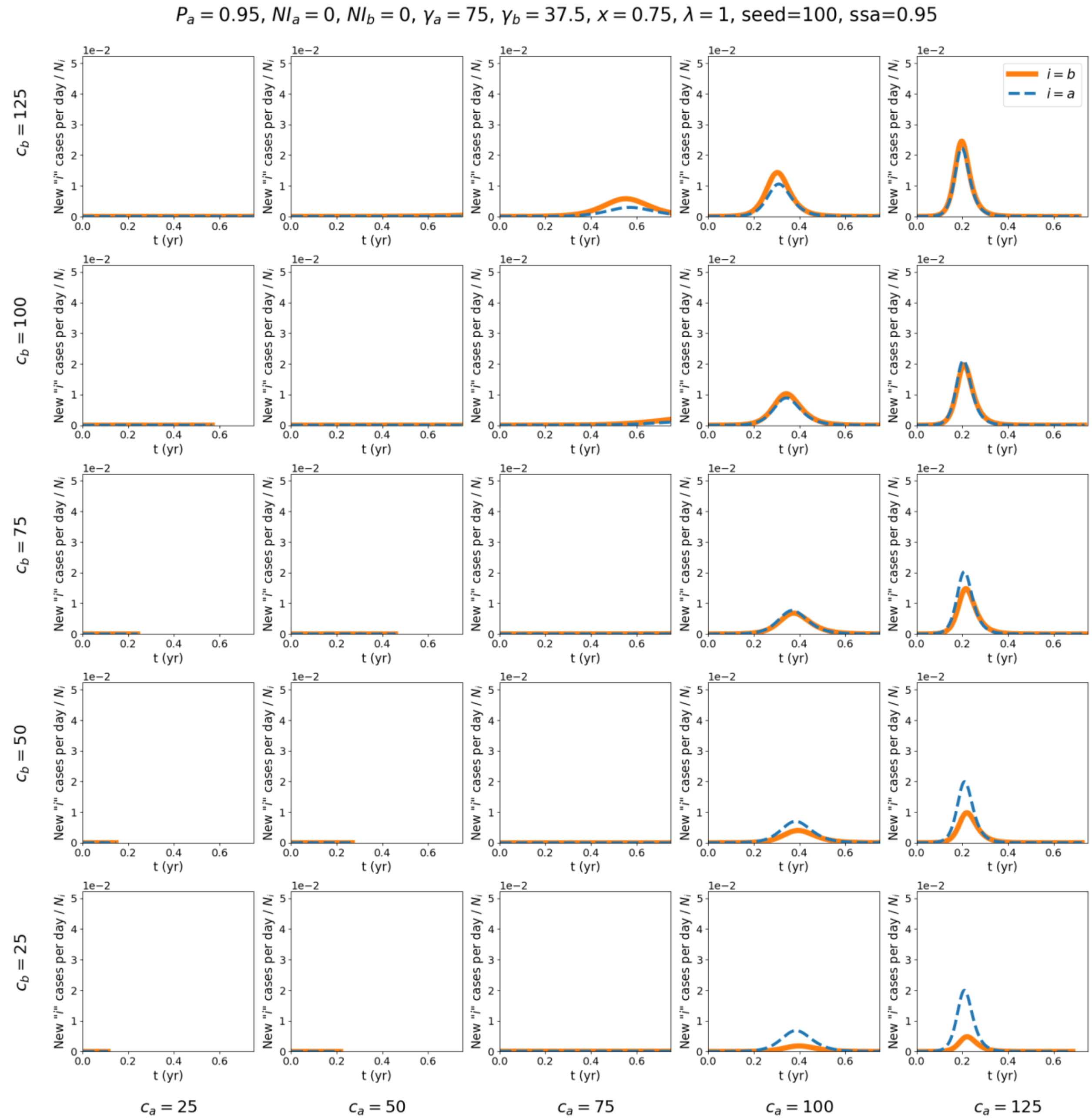

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$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2
$i = b$
$i = a$

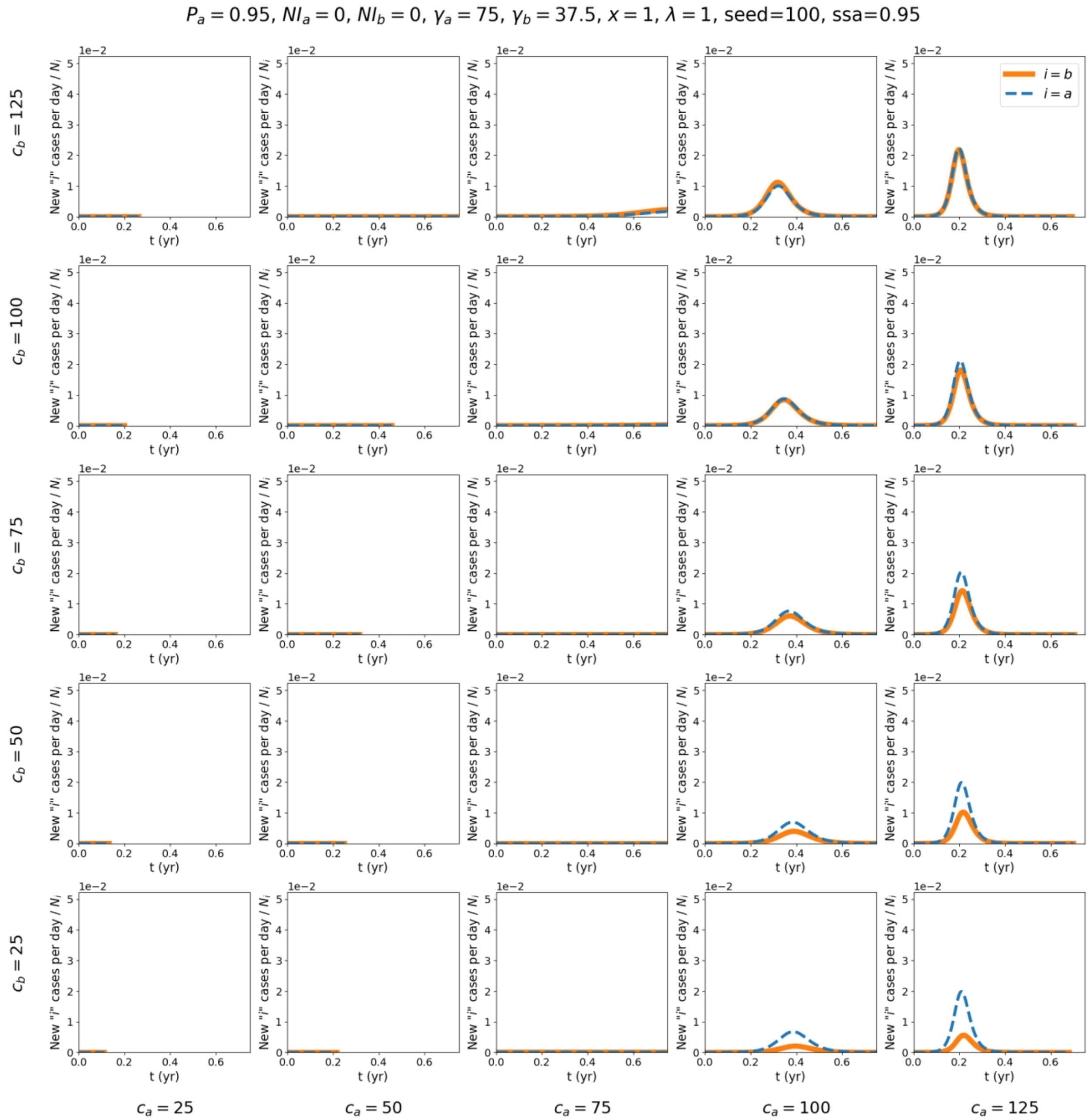

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$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2
$i = b$
$i = a$

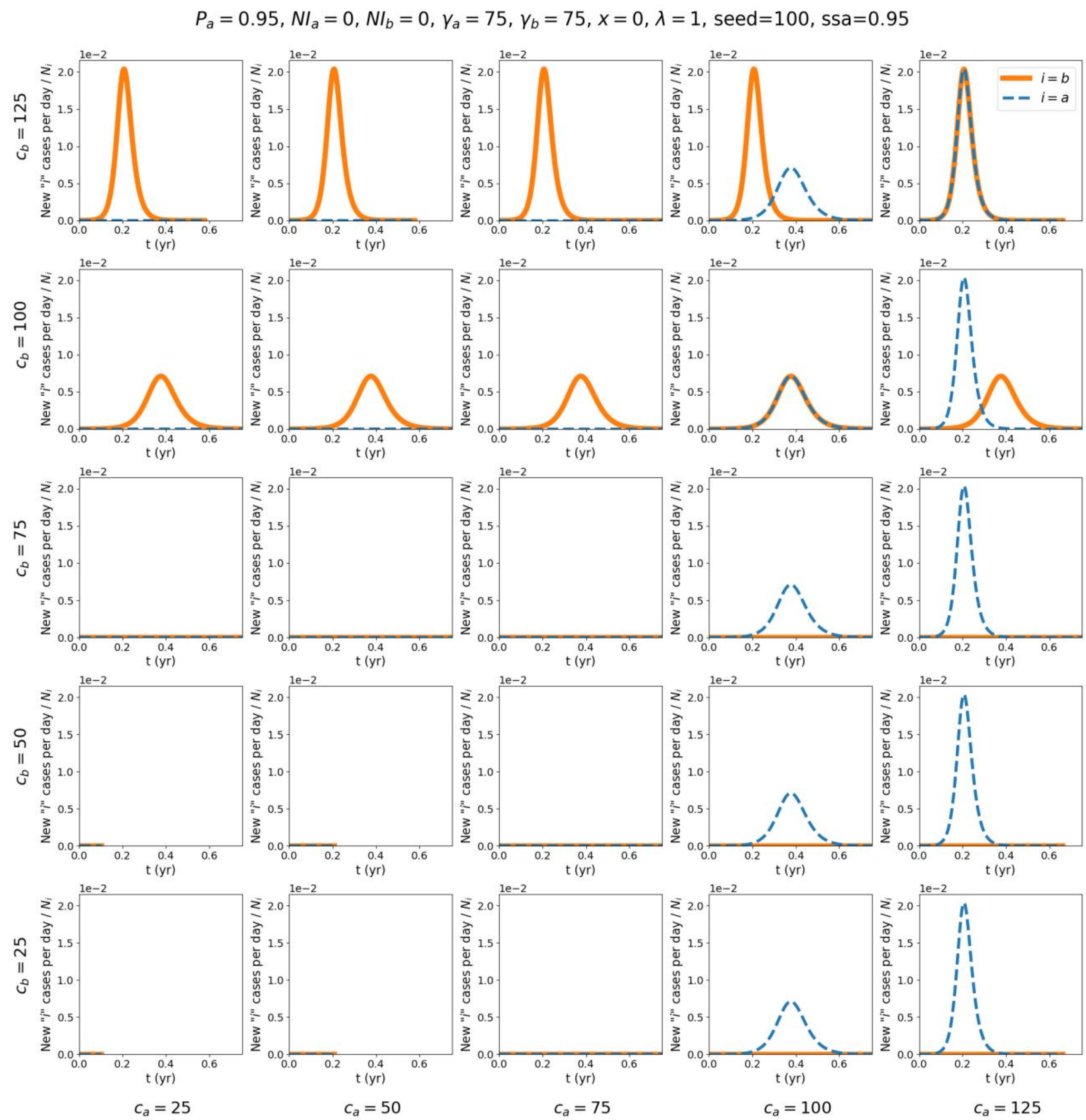

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$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2
$i = b$
$i = a$

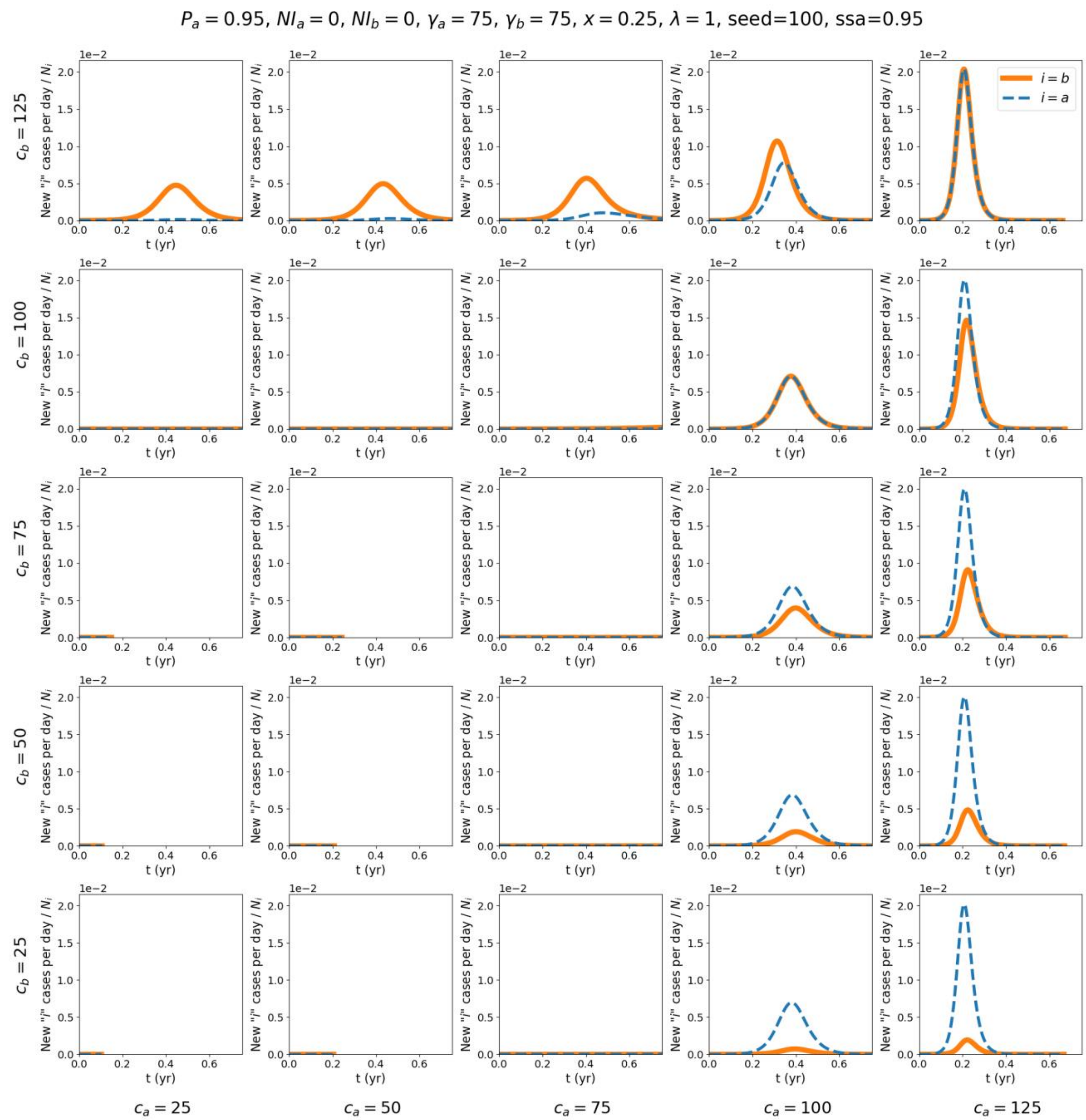

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$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2
$i = b$
$i = a$

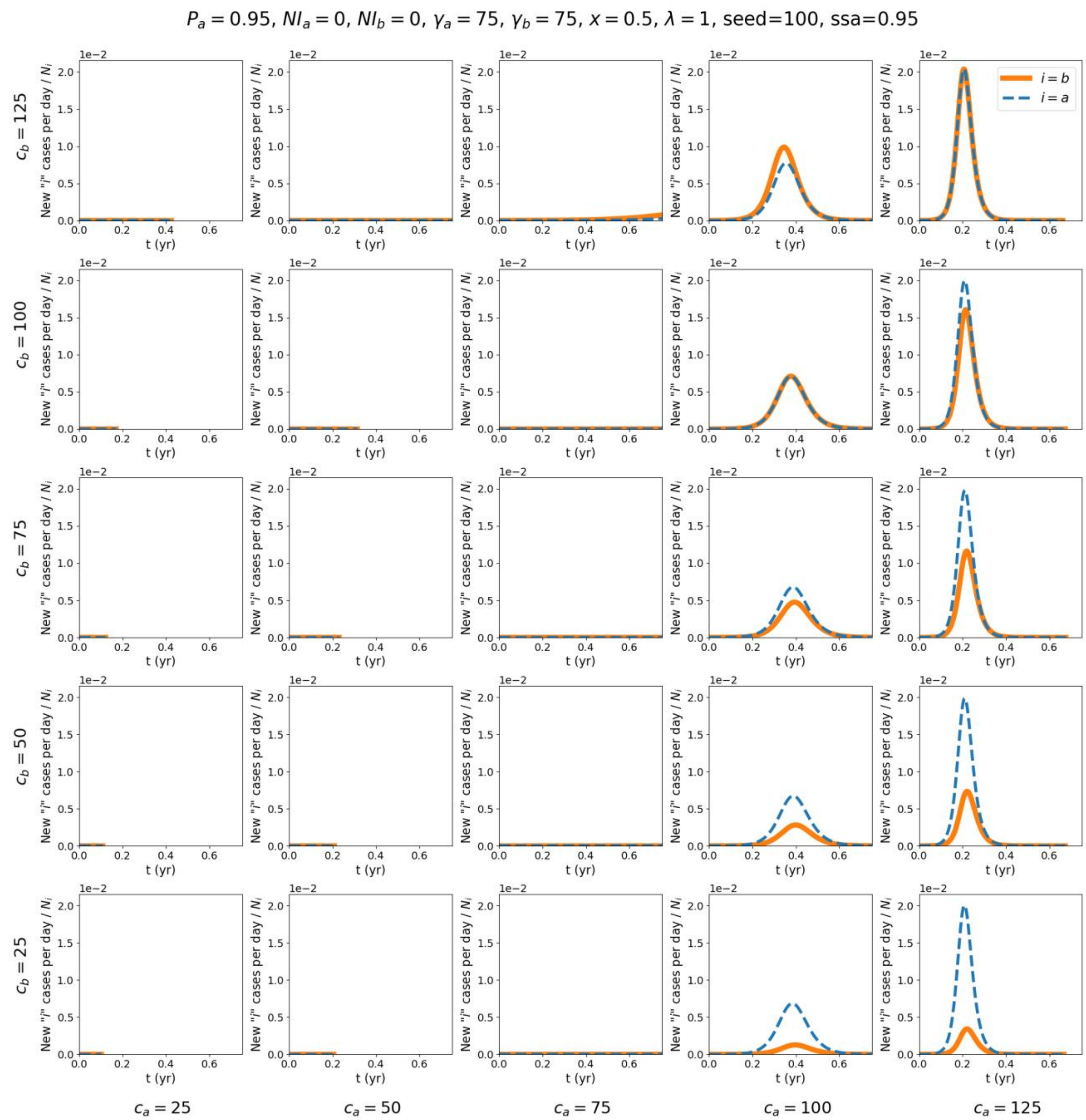
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$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 37.5$, $x = 0.75$, $\lambda = 1$, seed=100, ssa=0.95
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$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2
$i = b$
$i = a$

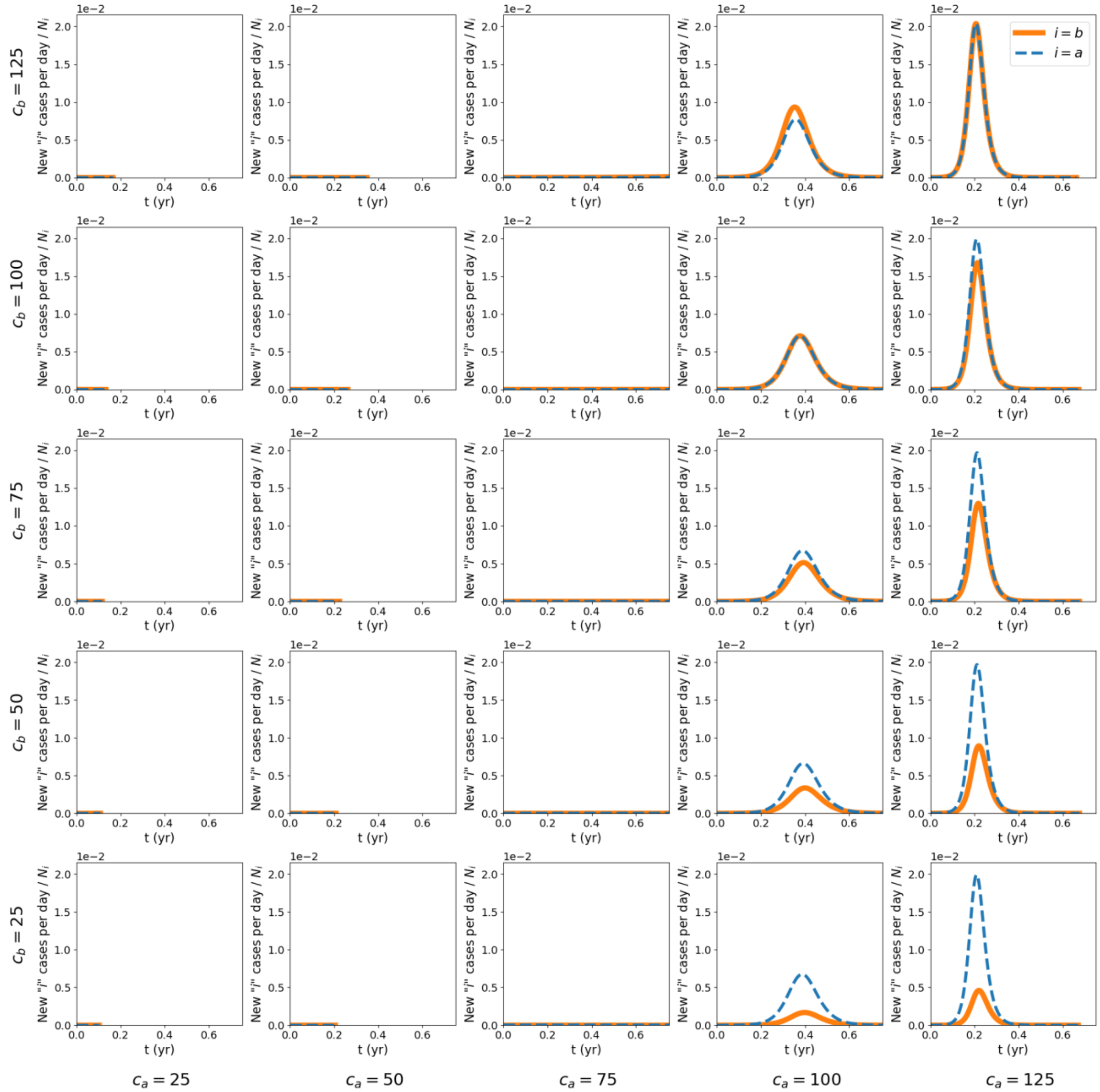

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$c_b = 100$
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$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2
$i = b$
$i = a$

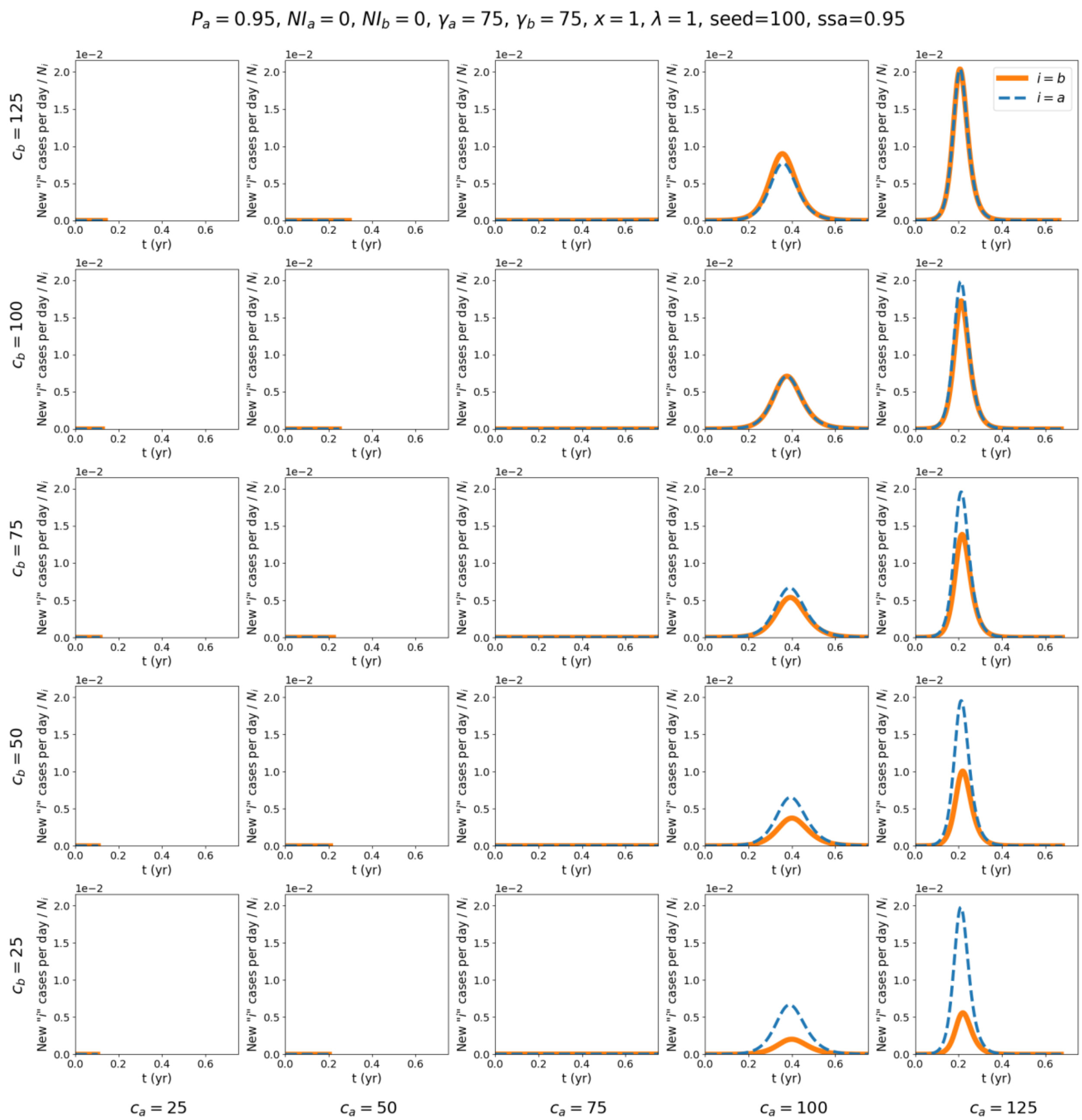

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$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
$i = b$
$i = a$
New "i" cases per day / N_i
t (yr)
1e−2


$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 75$, $x = 0.25$, $\lambda = 1$, seed=100, ssa=0.95
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$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2
$i = b$
$i = a$


$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 75$, $x = 0.5$, $\lambda = 1$, seed=100, ssa=0.95
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2
$i = b$
$i = a$

$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 75$, $x = 0.75$, $\lambda = 1$, seed=100, ssa=0.95




$P_a = 0.95$, $NI_a = 0$, $NI_b = 0$, $\gamma_a = 75$, $\gamma_b = 75$, $x = 1$, $\lambda = 1$, seed=100, ssa=0.95
$c_b = 125$
$c_b = 100$
$c_b = 75$
$c_b = 50$
$c_b = 25$
$c_a = 25$
$c_a = 50$
$c_a = 75$
$c_a = 100$
$c_a = 125$
New "i" cases per day / N_i
t (yr)
1e−2
$i = b$
$i = a$

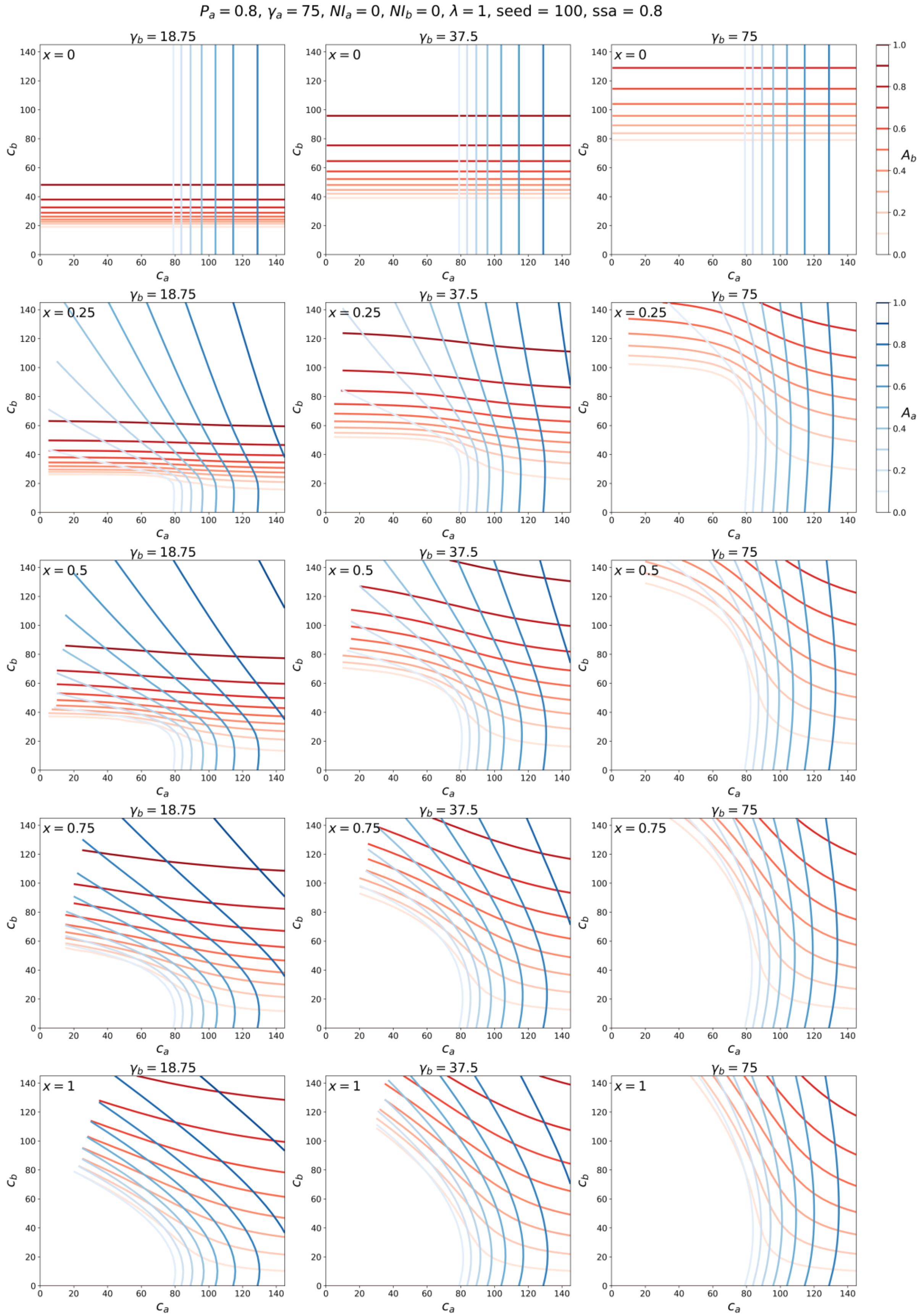
Appendix B: Results for P_a = 0.8 and P_a = 0.6

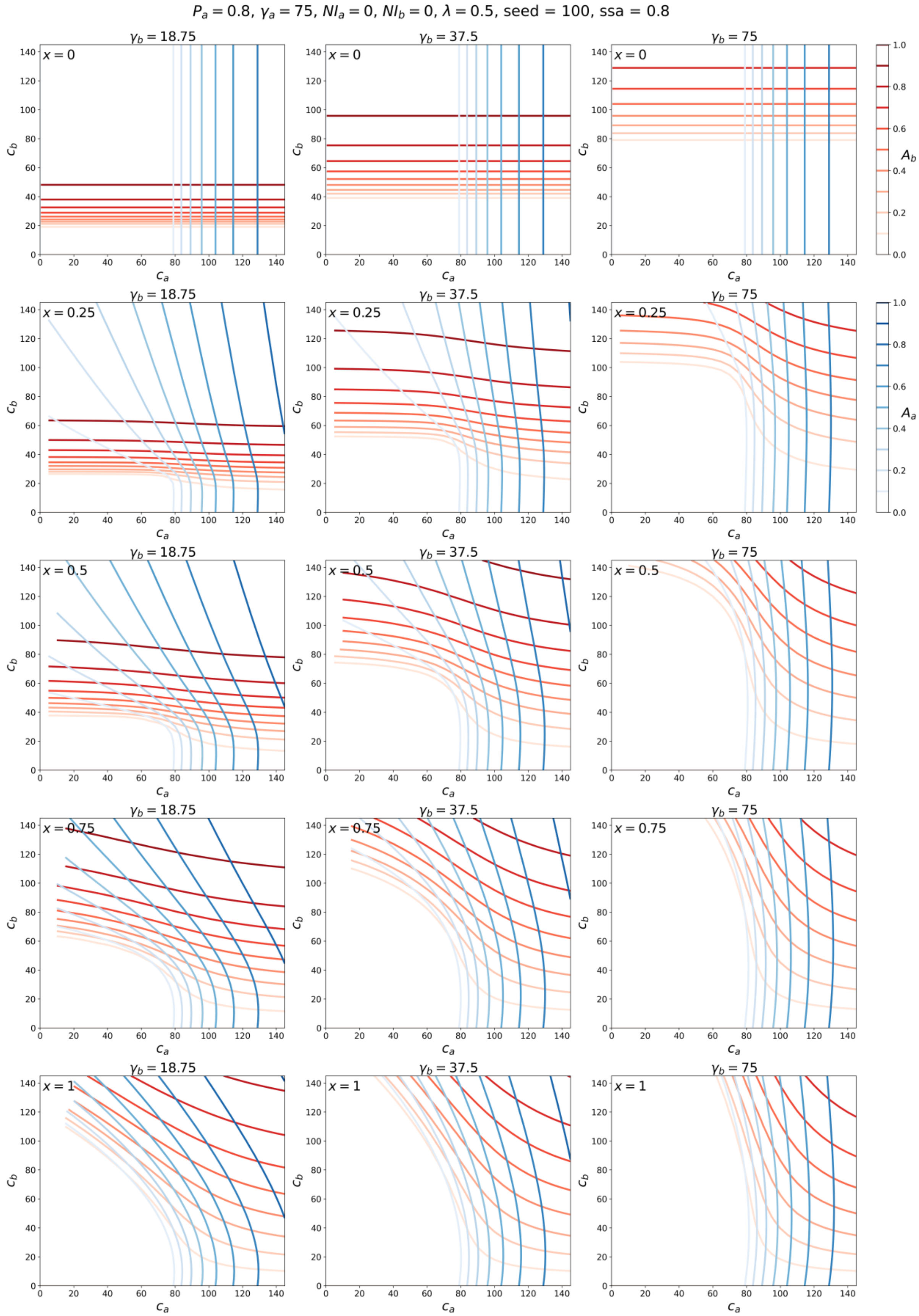
This Appendix shows attack-rate contour maps for two different values of P_a, for the same values of c_a, c_b, and x used in the main text and elsewhere in the Appendices.

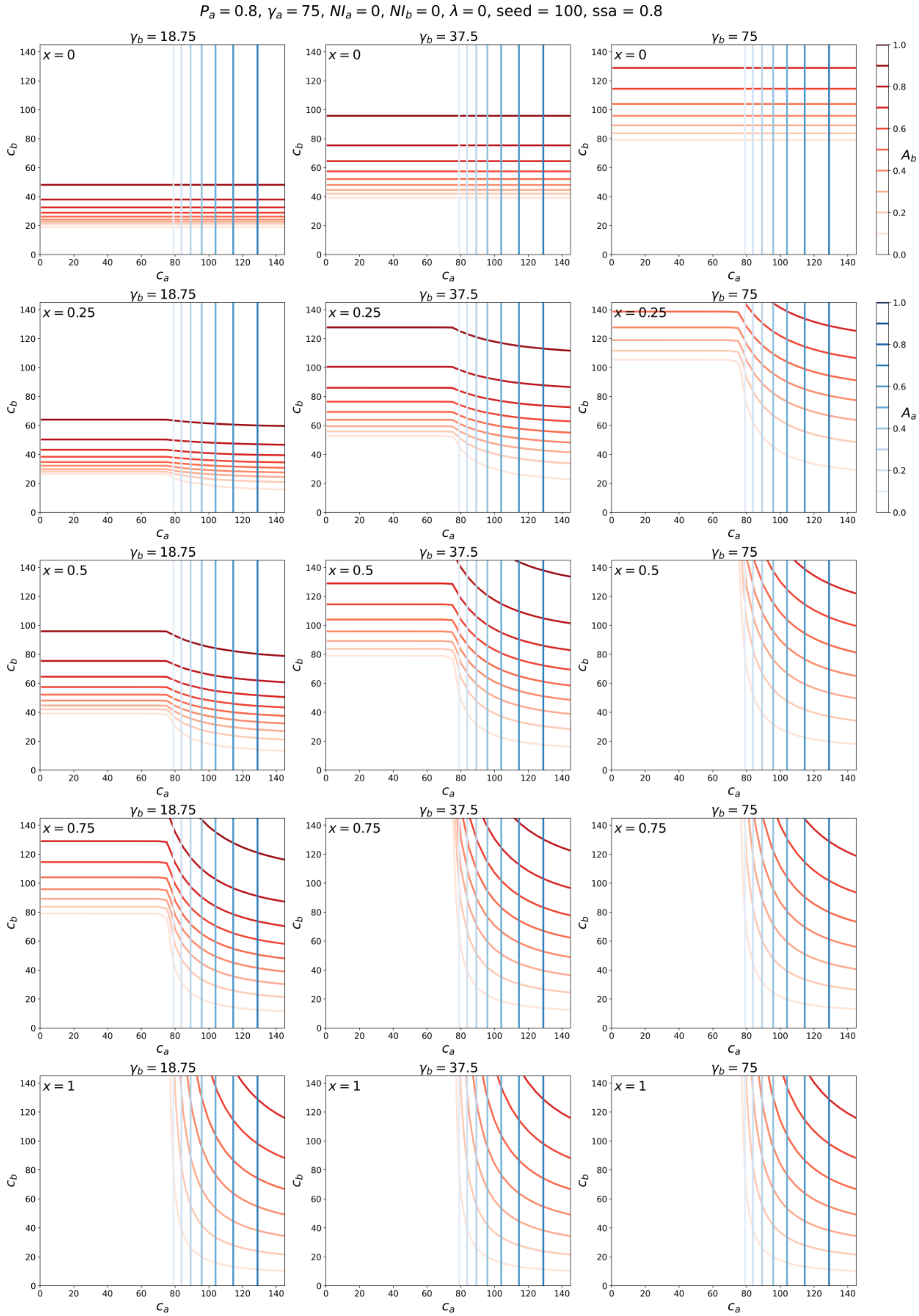
Note that Eqs. 2 and 4 of the main text impose constraints on the c_{ij}. In some of the contour maps shown in Appendix B and Appendix C, the contour lines end abruptly at points in the (c_a, c_b) plane where these constraints are reached.

For example, for $P_a = 0.8$, $x = 1$, and $\gamma_b = 18.75$, the point ($c_a = 20$, $c_b = 100$) is unphysical, because $x = 1$ implies that $c_{ba} = c_b = 100$ (see Eq. 4, main text), and $\lambda = 1$, $P_a = 0.8$, and $c_{ba} = 100$ are such (from Eq. 2, main text) that $c_{ab} = \lambda c_{ba}(1-P_a)/P_a = 25 > c_a$ which is unphysical, since $c_a = c_{aa} + c_{ab}$ and both $c_{aa} \geq 0$ and $c_{ab} \geq 0$. Accordingly, the contour lines in the contour map for $P_a = 0.8$, $x = 1$, and $\gamma_b = 18.75$ (lower-left panel in the first figure in section B.1, below) end before the unphysical point ($c_a = 20$, $c_b = 100$).

B.1: Attack-rate contour maps for different values of λ and for P_a = 0.8

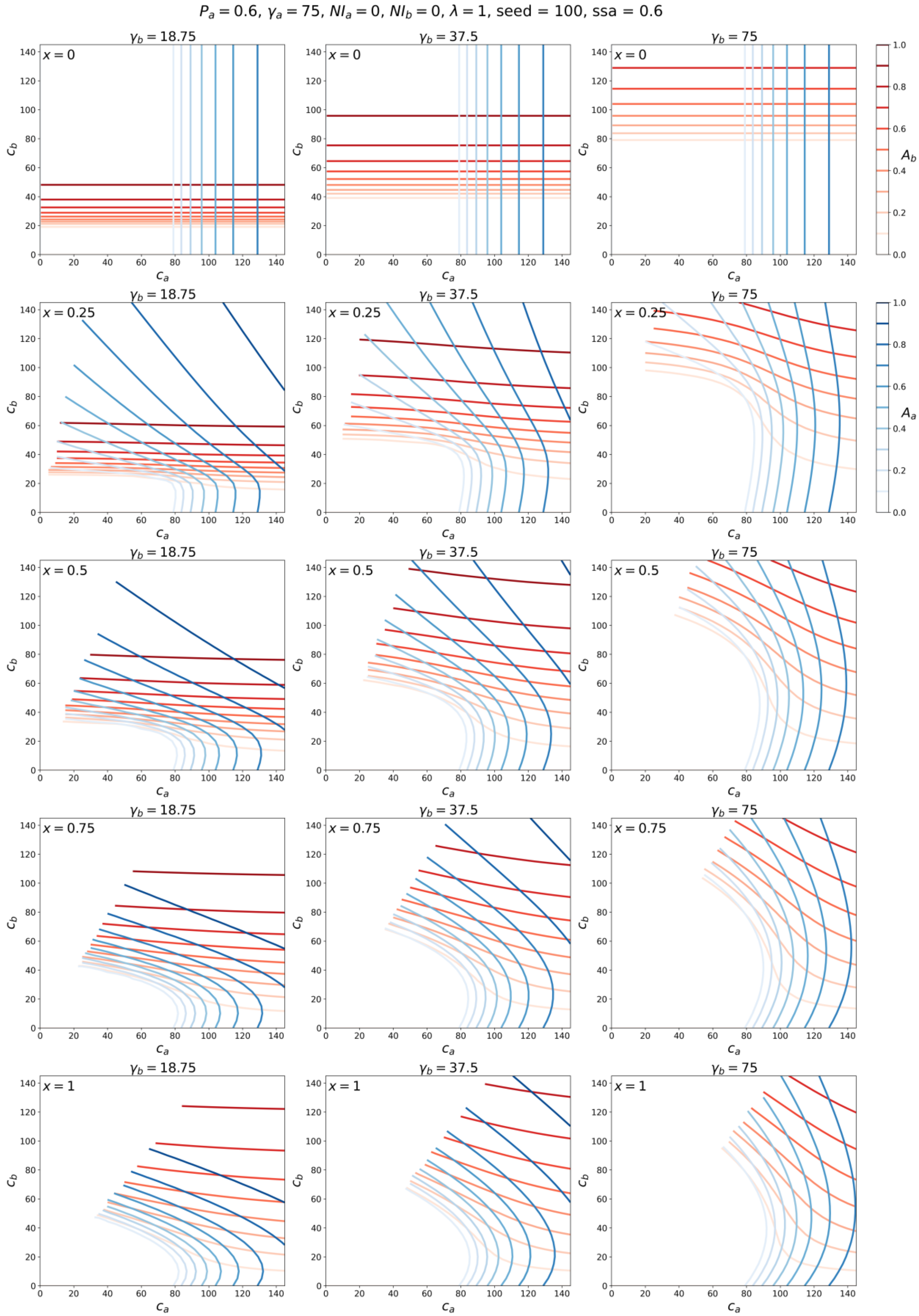

$P_a = 0.8$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 1$, seed = 100, ssa = 0.8
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a


$P_a = 0.8$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0.5$, seed = 100, ssa = 0.8
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

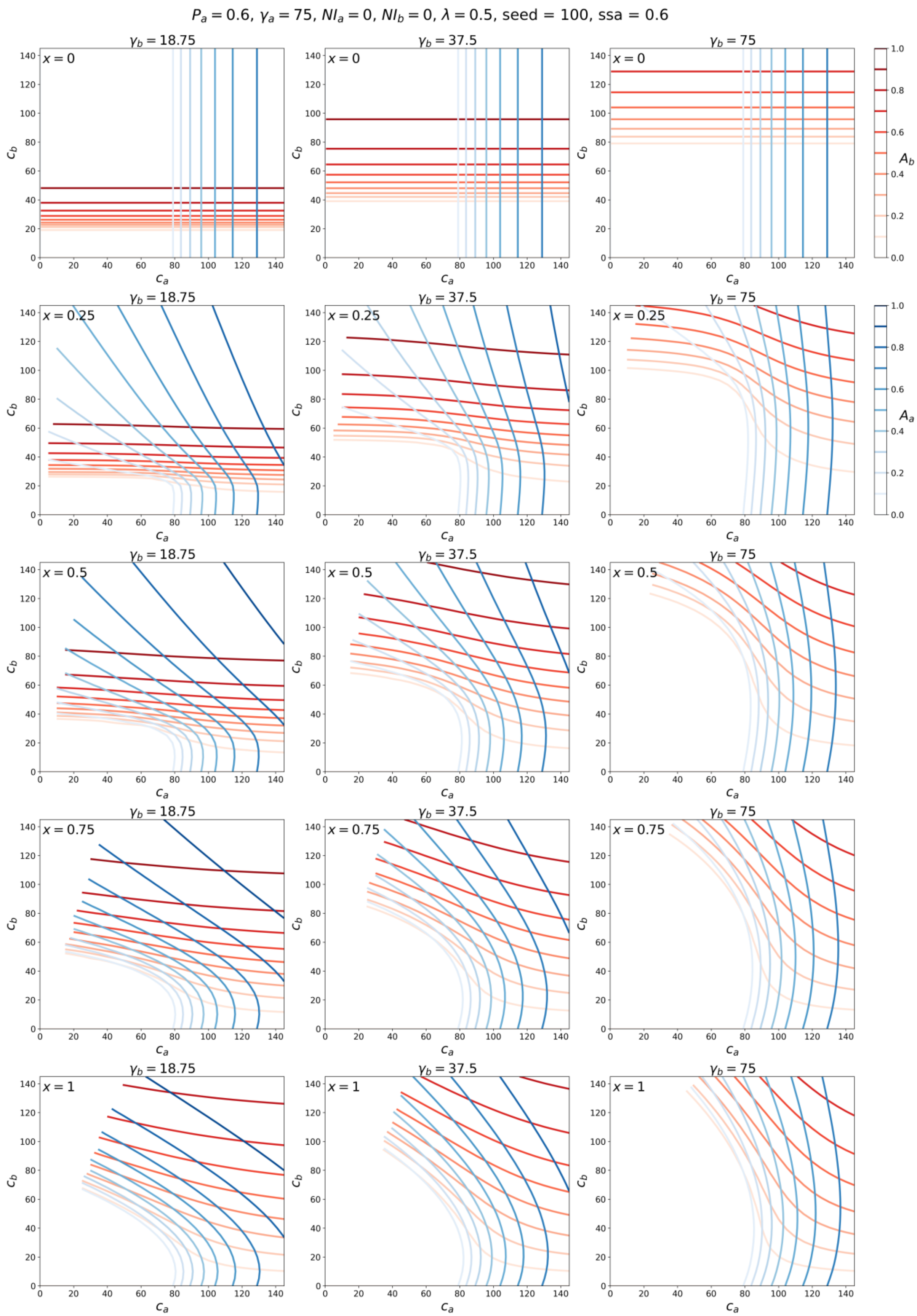

$P_a = 0.8$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0$, seed = 100, ssa = 0.8
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

98

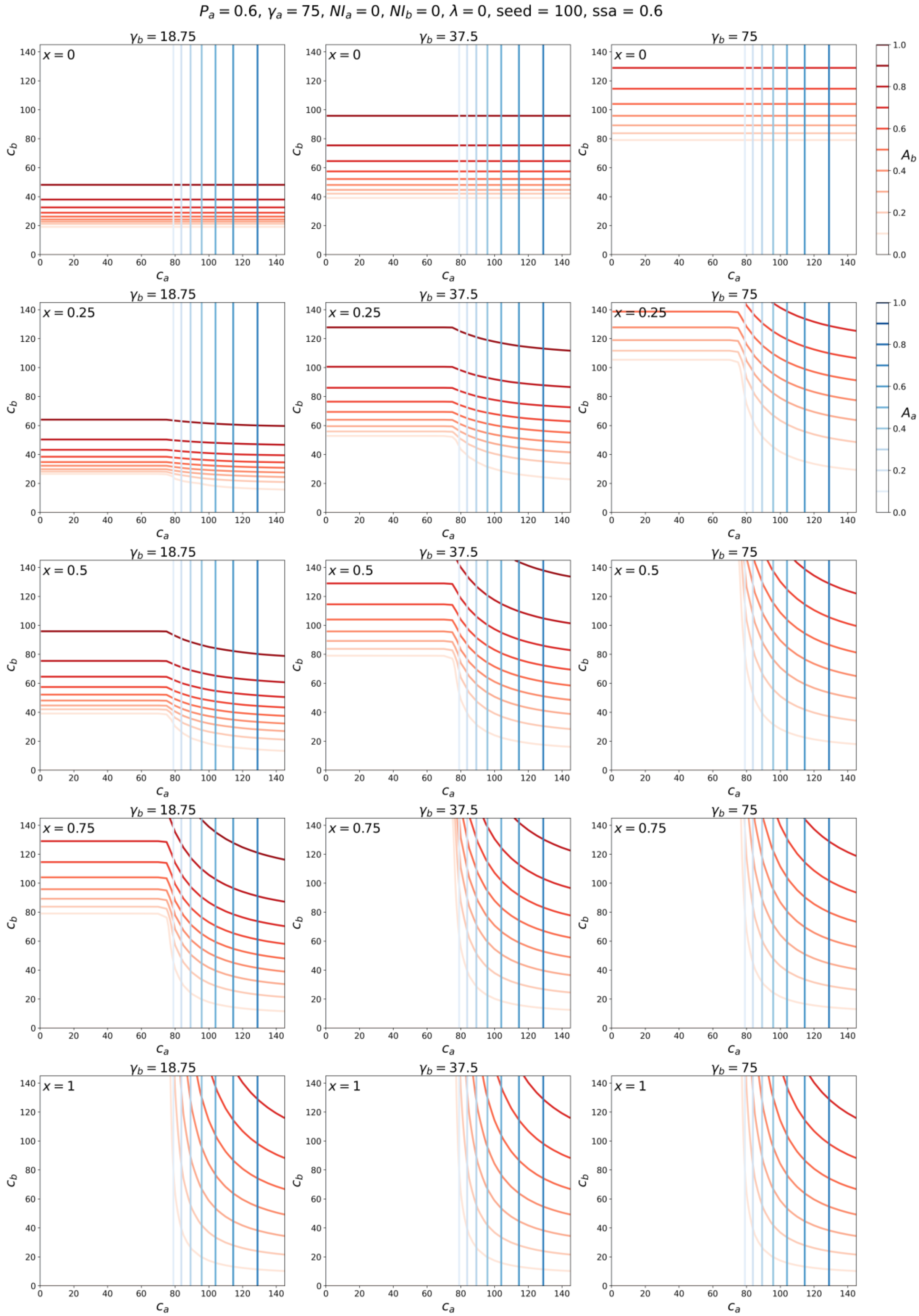
B.2: Attack-rate contour maps for different values of λ and for P_a = 0.6


$P_a = 0.6$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 1$, seed = 100, ssa = 0.6
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

100


$P_a = 0.6$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0.5$, seed = 100, ssa = 0.6
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

101

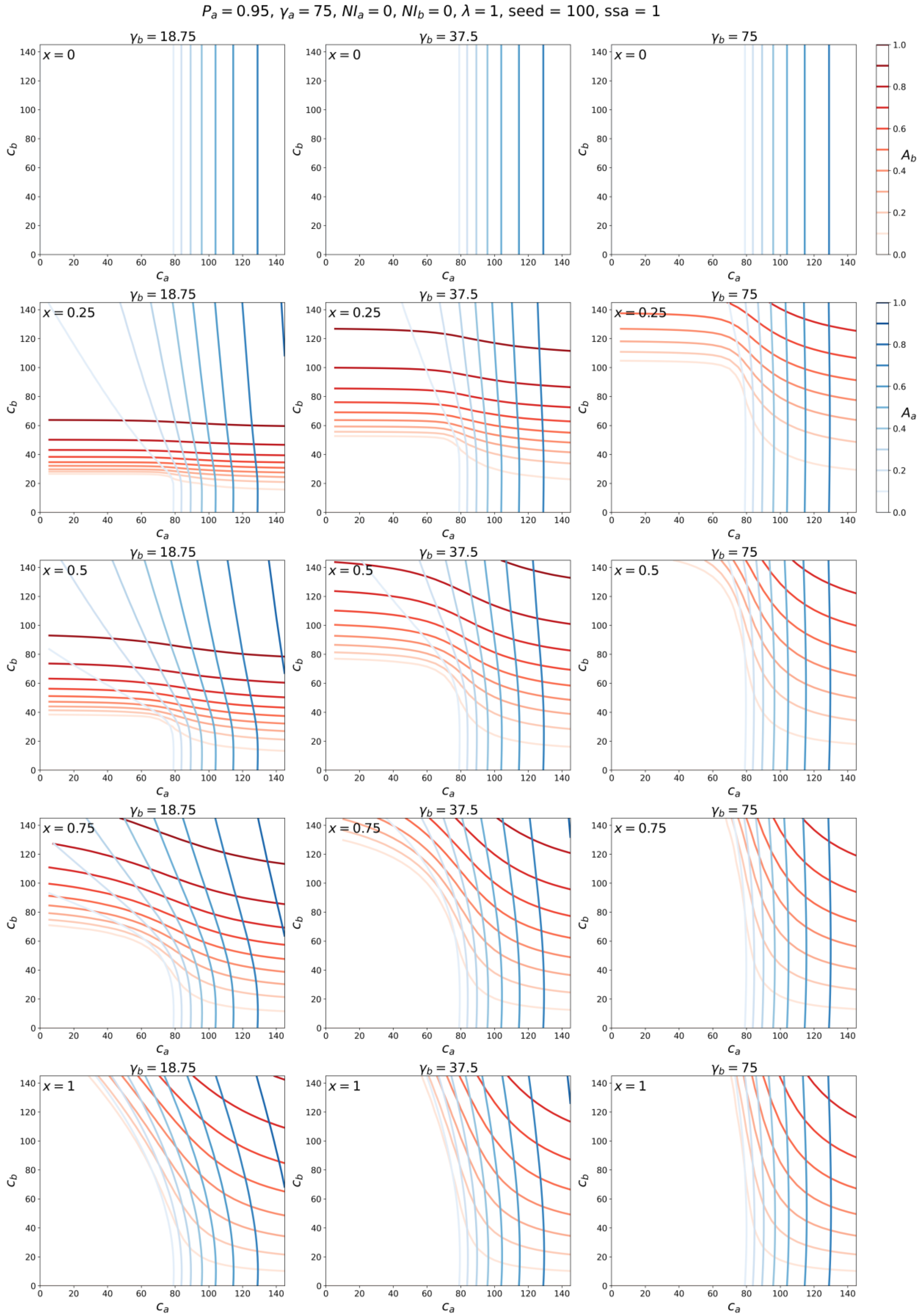

$P_a = 0.6$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0$, seed = 100, ssa = 0.6
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

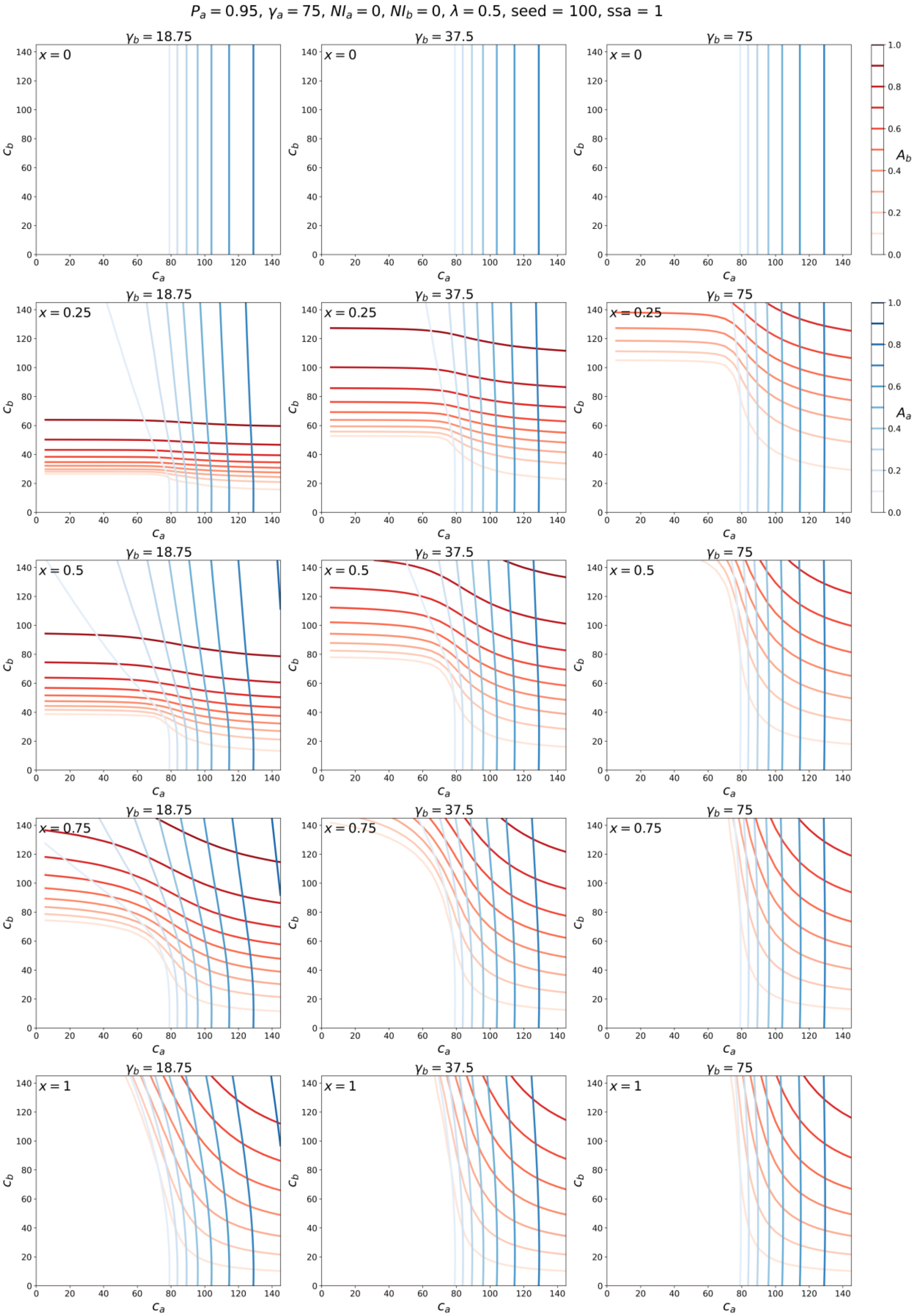
Appendix C: Varying seed distribution

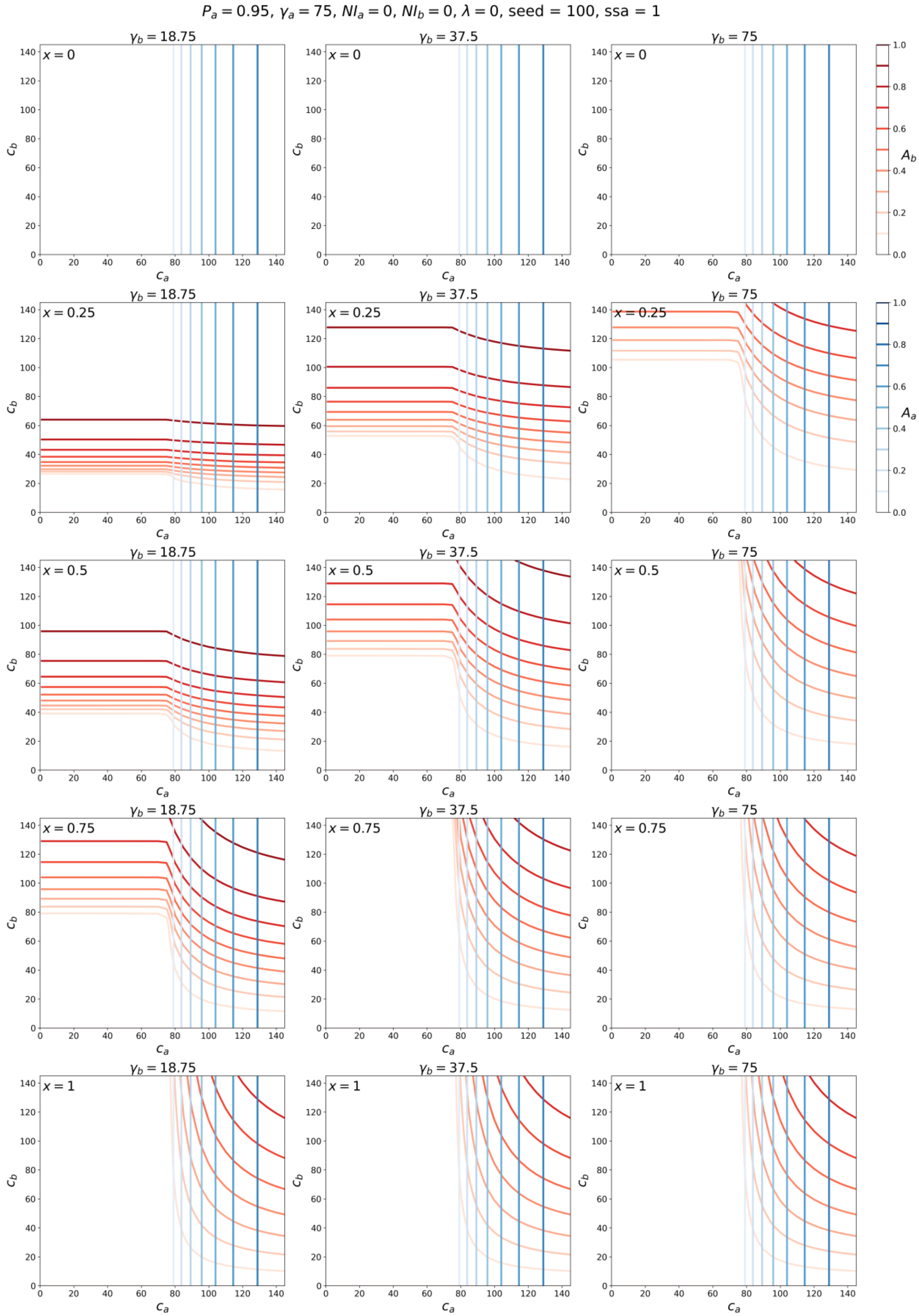
The figures in Appendix C show contour maps for P_a = 0.95, P_a = 0.8, and P_a = 0.6, with the initial 100 infected "seed" individuals placed entirely in the *a* population (ssa = 1). That is, none of the *b* individuals are initially infected, in the simulations shown in Appendix C.

As can be seen from the figures below, placing all "seed" individuals in the *a* group has no effect on the resulting attack rates as functions of c_a, c_b, and x, except for the trivial case of $x = 0$, in which it is impossible for any *b* person to become infected, since $x = 0$ means that $c_{ba} = 0$.

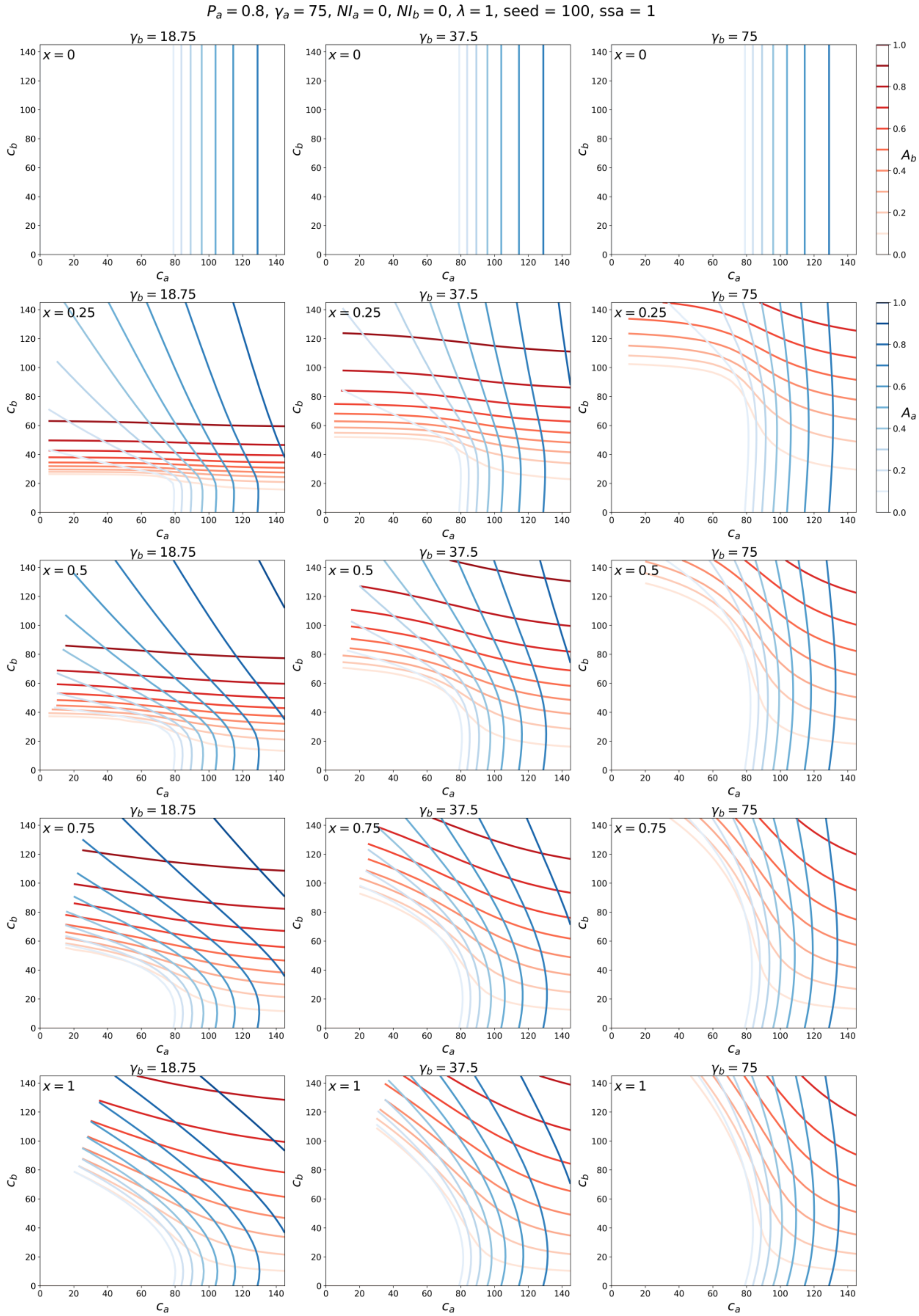
C.1: Attack-rate contour maps for different values of λ, for P_a = 0.95, seed = 100, and ssa = 1

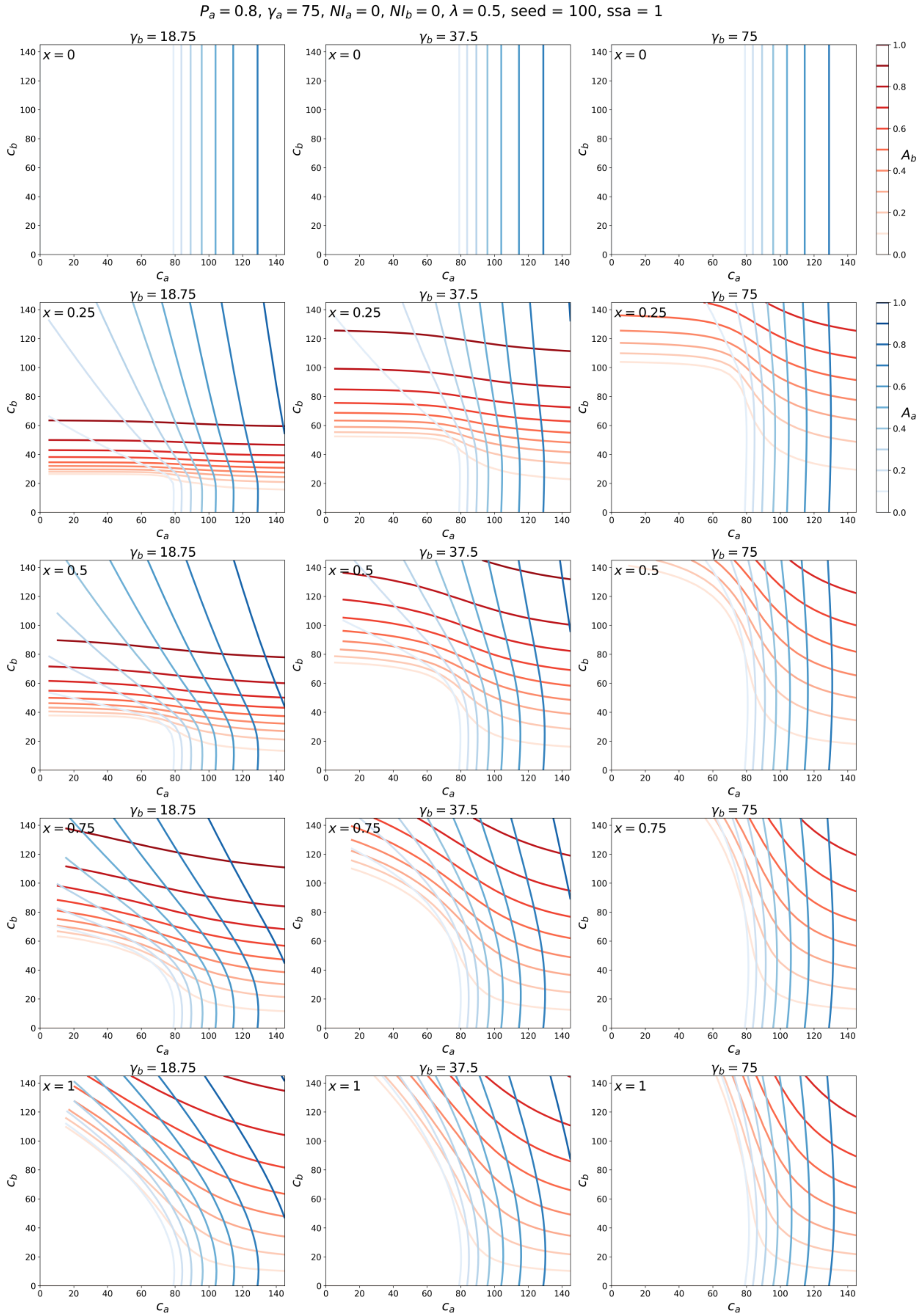

$P_a = 0.95$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 1$, seed = 100, ssa = 1
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

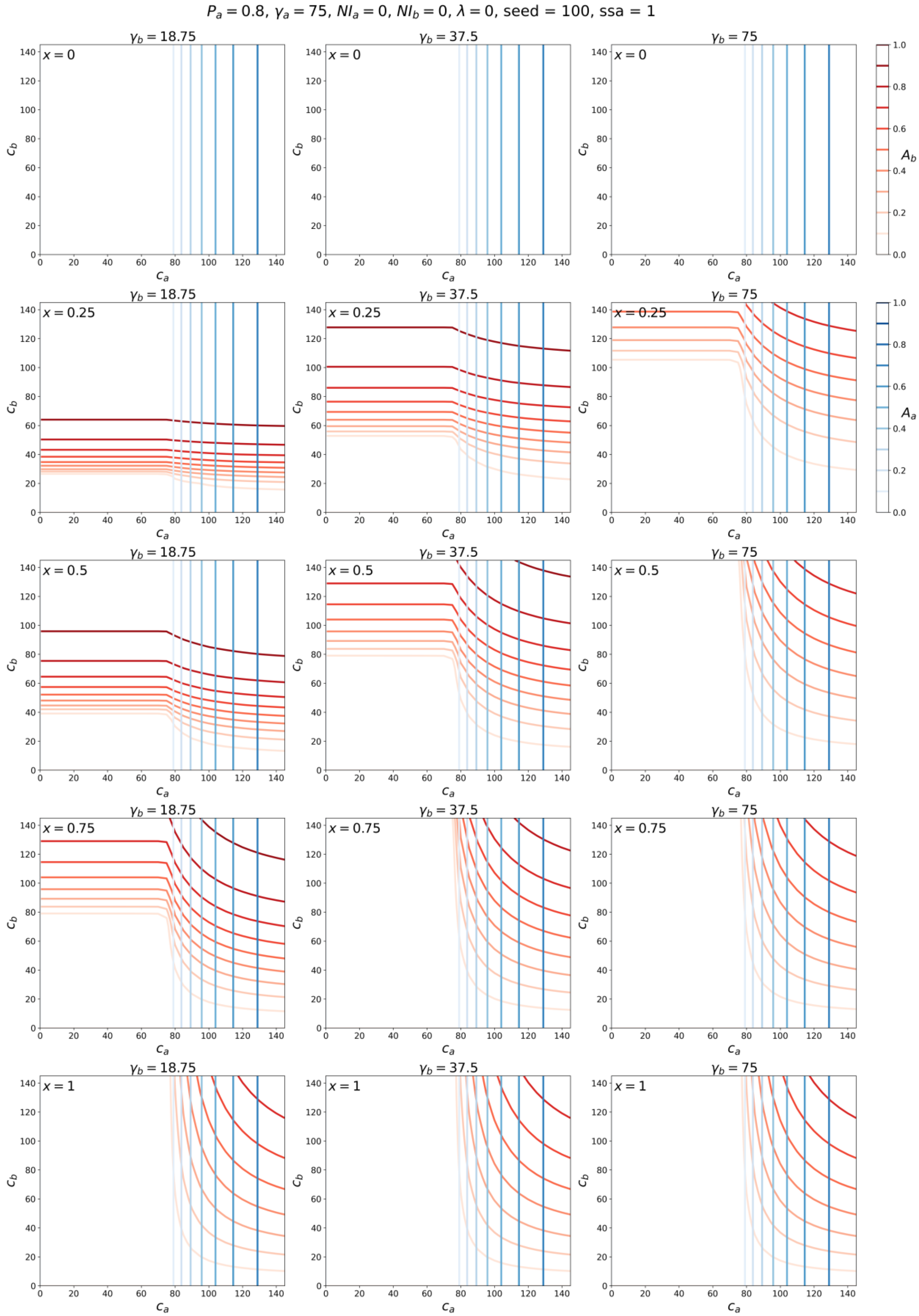

$P_a = 0.95$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0.5$, seed = 100, ssa = 1
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a


$P_a = 0.95$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0$, seed = 100, ssa = 1
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

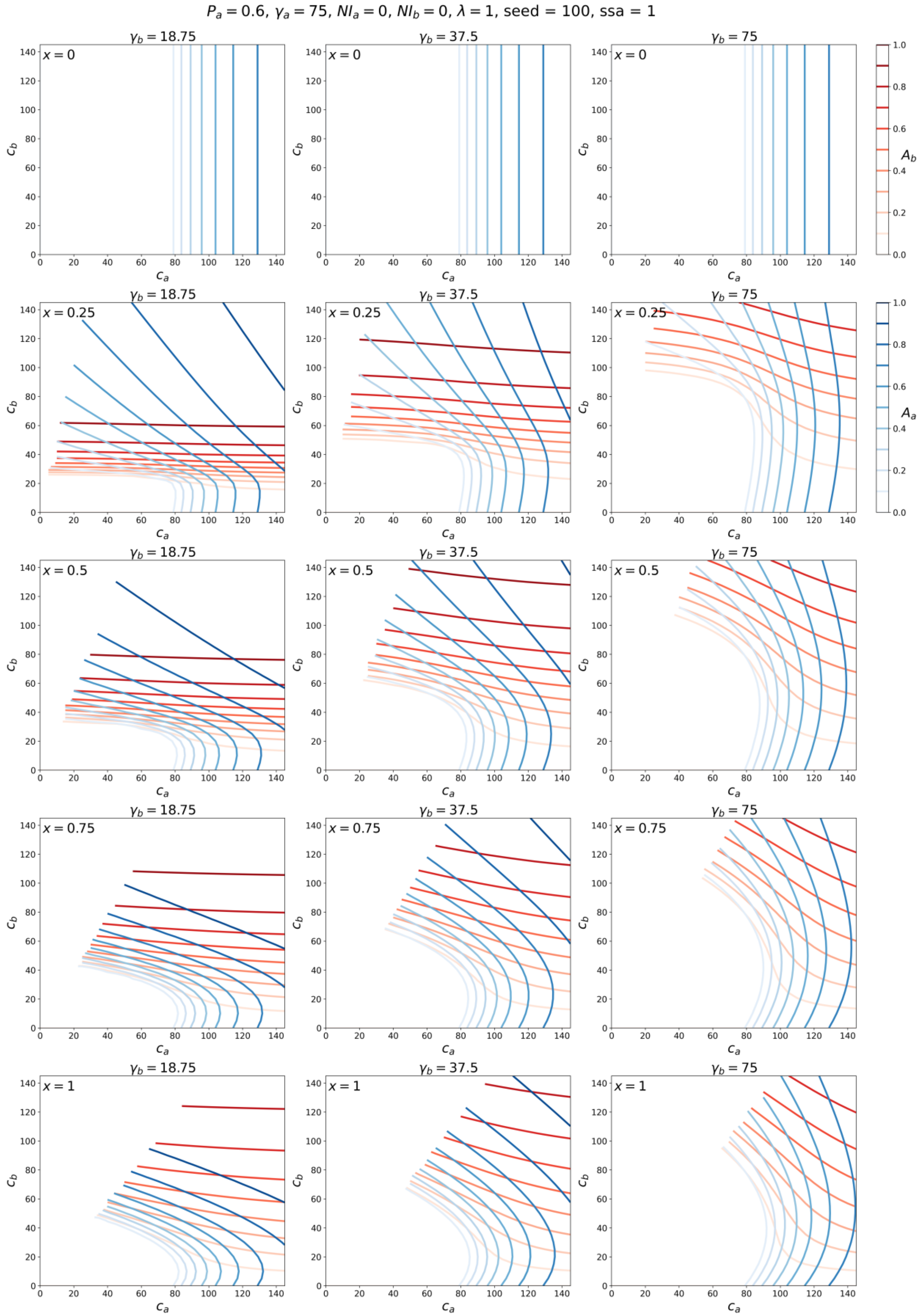
C.2: Attack-rate contour maps for different values of λ, for P_a = 0.8, seed = 100, and ssa = 1

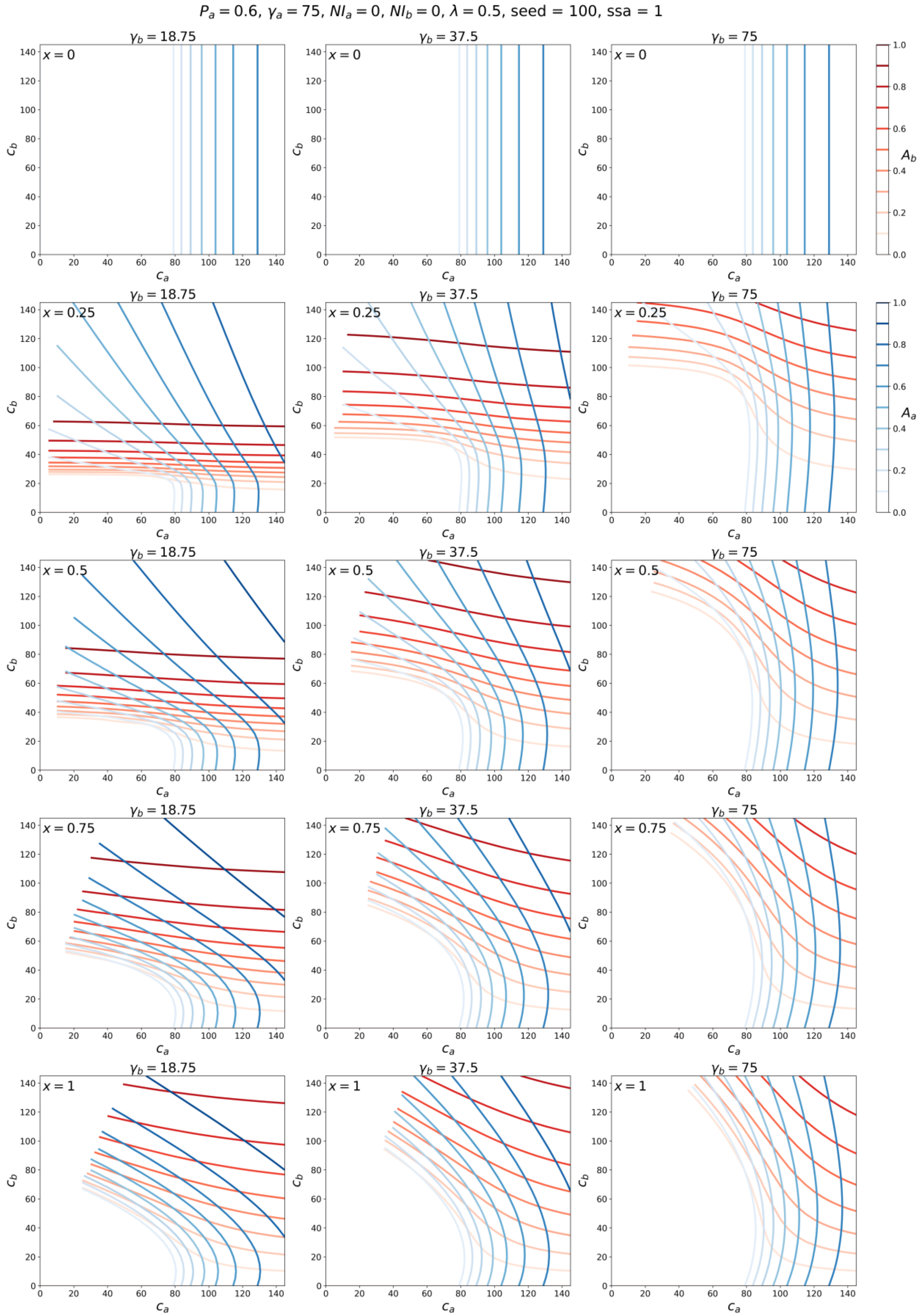

$P_a = 0.8$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 1$, seed = 100, ssa = 1
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

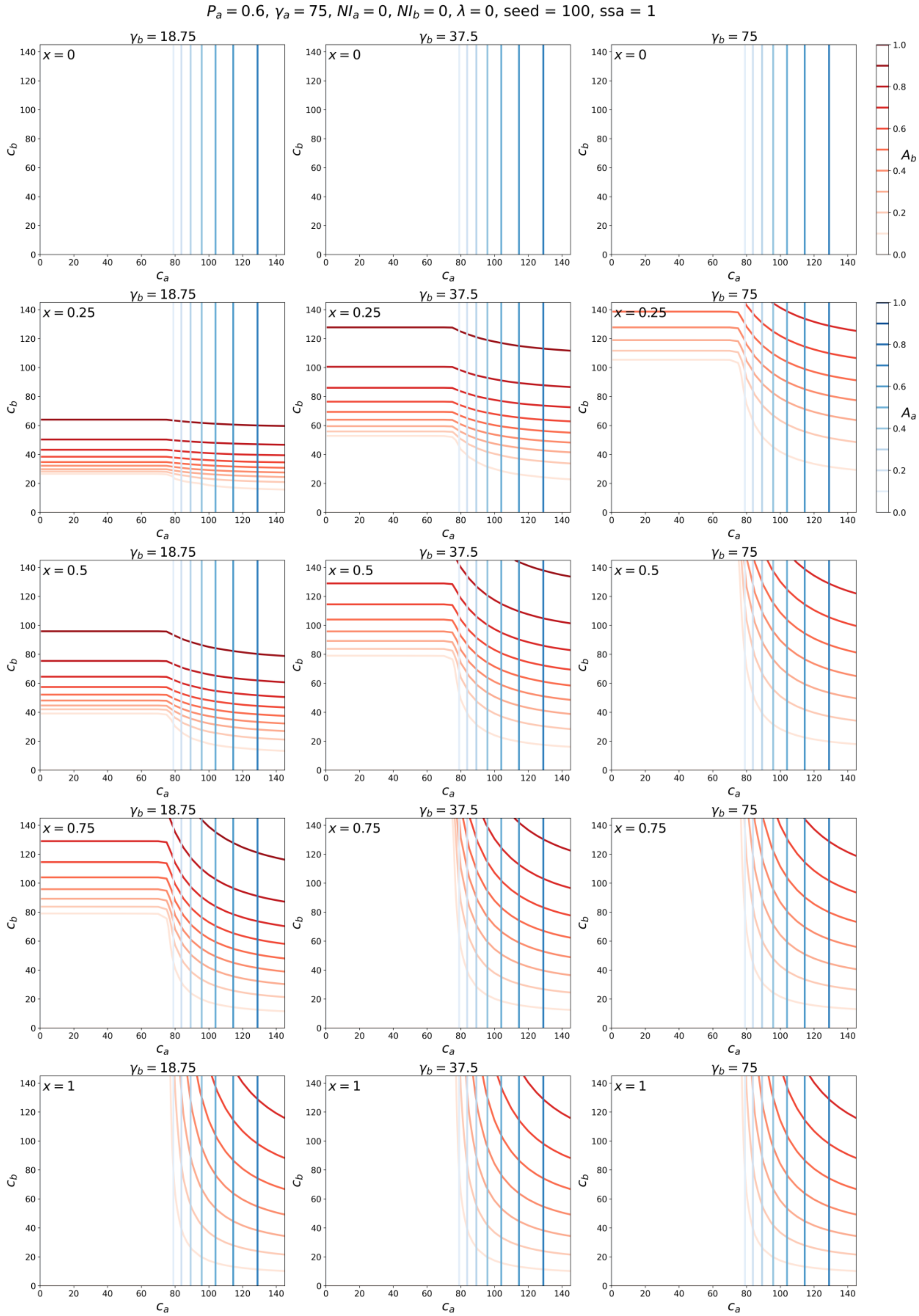

$P_a = 0.8$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0.5$, seed = 100, ssa = 1
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a


$P_a = 0.8$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0$, seed = 100, ssa = 1
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

C.3: Attack-rate contour maps for different values of λ, for P_a = 0.6, seed = 100, and ssa = 1


$P_a = 0.6$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 1$, seed = 100, ssa = 1
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

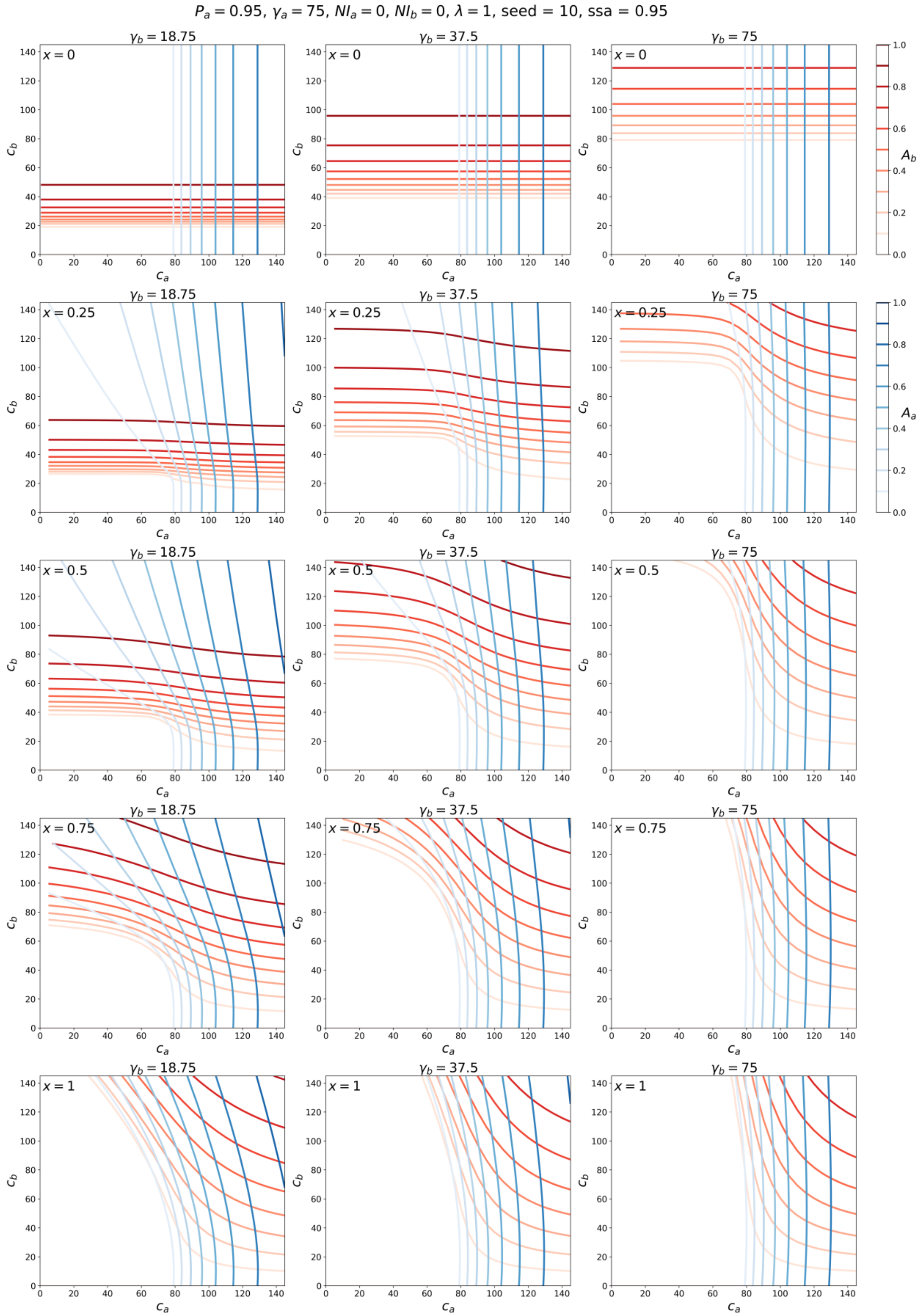

$P_a = 0.6$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0.5$, seed = 100, ssa = 1
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

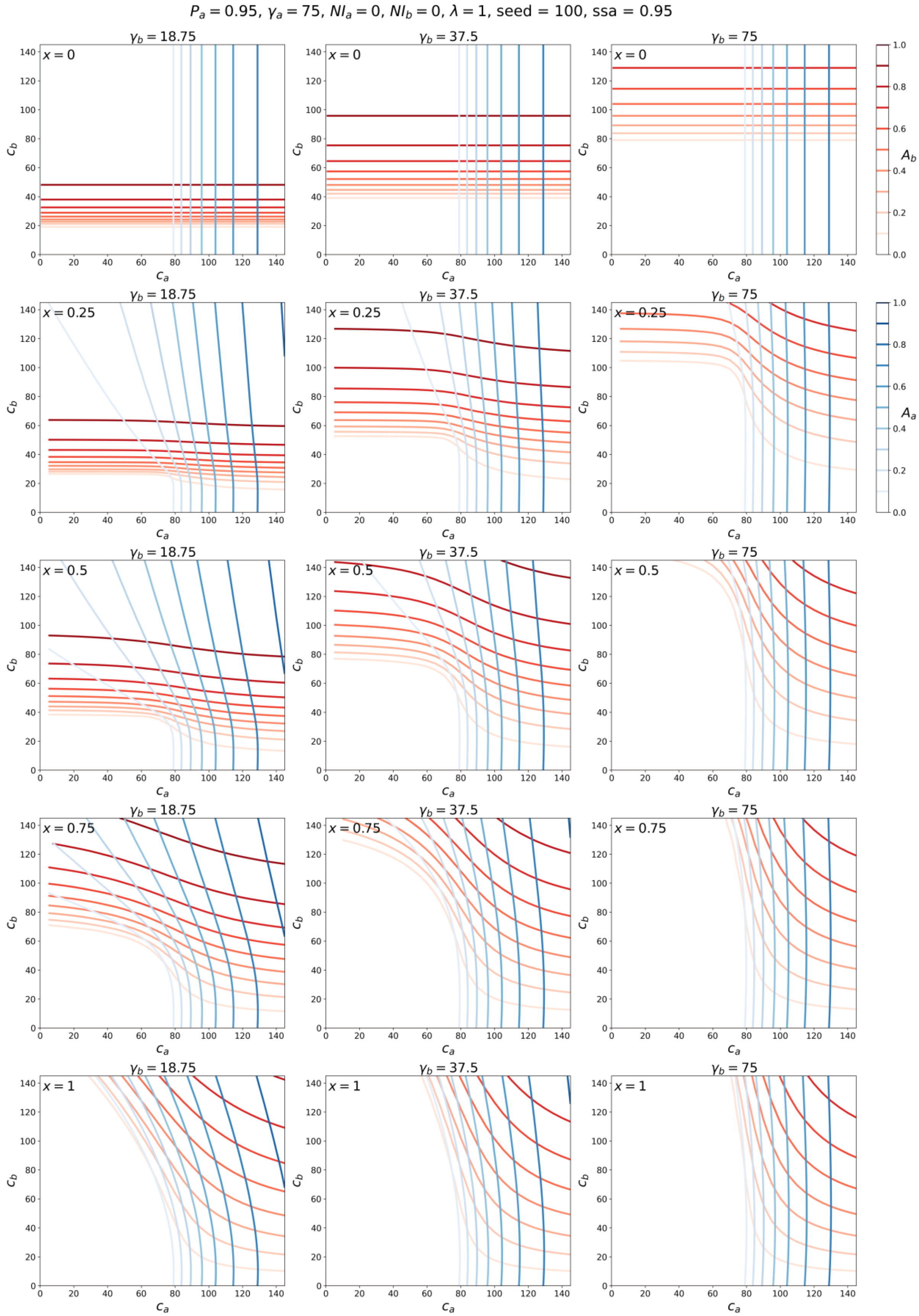

$P_a = 0.6$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 0$, seed = 100, ssa = 1
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

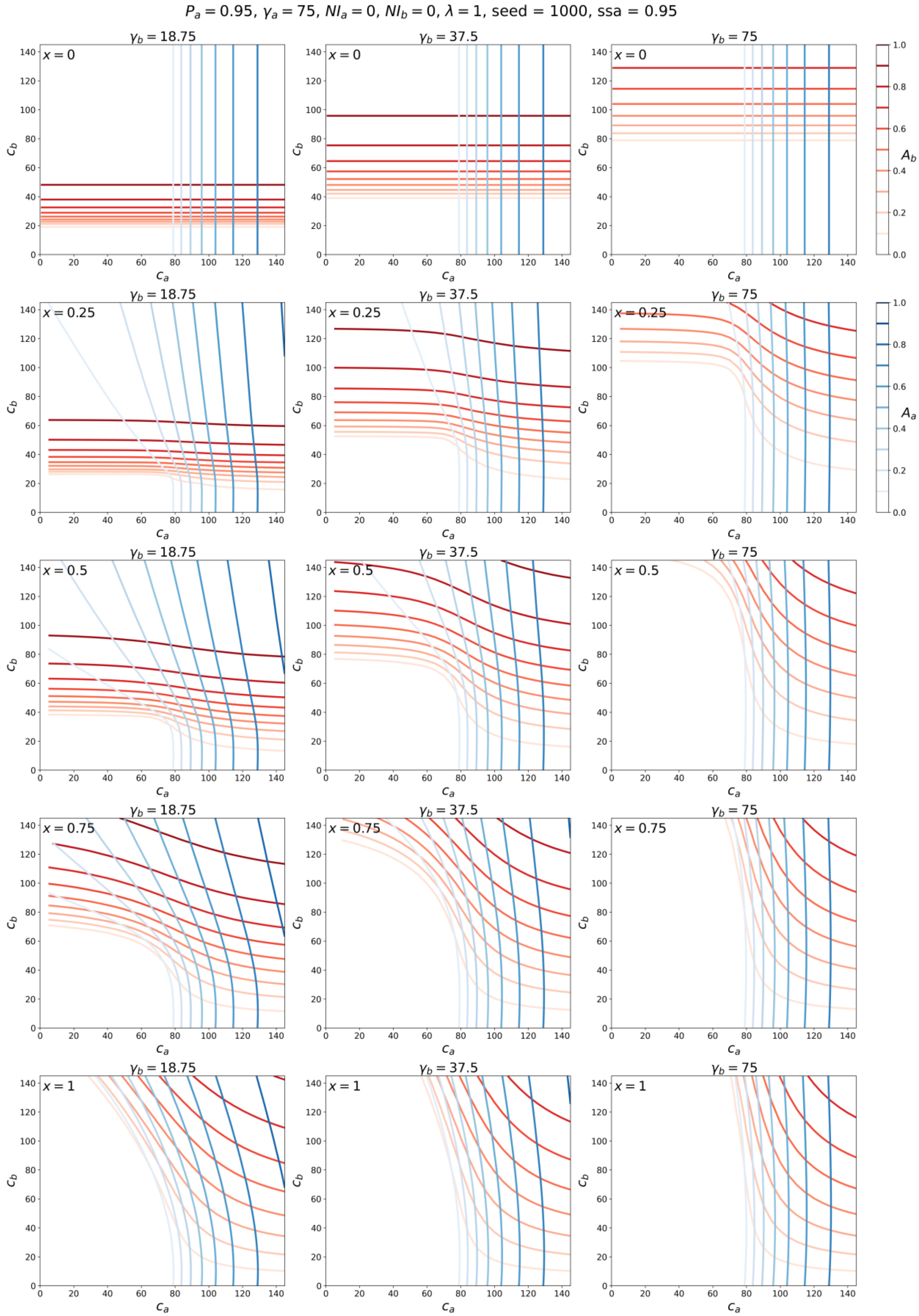
115

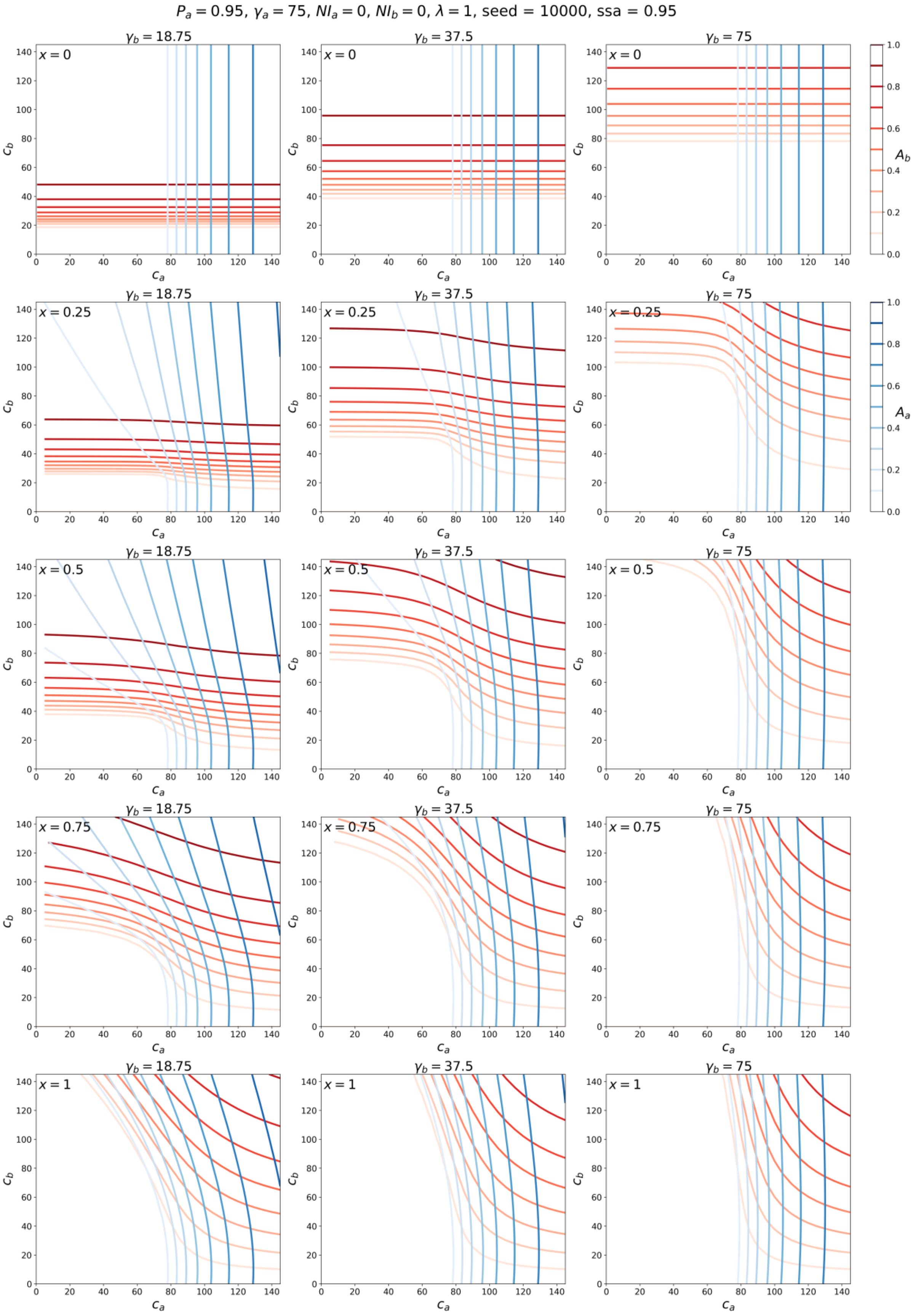
Appendix D: Varying seed magnitude

This Appendix contains attack-rate contour maps for the parameters used in the main text figures, but for different magnitudes of the initial seed number of infected individuals (parameter "seed"). As can be seen, changing the seed magnitude does not change the attack-rate results.


$P_a = 0.95$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 1$, seed = 10, ssa = 0.95
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a


$P_a = 0.95$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 1$, seed = 100, ssa = 0.95
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a


$P_a = 0.95$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 1$, seed = 1000, ssa = 0.95
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a


$P_a = 0.95$, $\gamma_a = 75$, $NI_a = 0$, $NI_b = 0$, $\lambda = 1$, seed = 10000, ssa = 0.95
$\gamma_b = 18.75$
$\gamma_b = 37.5$
$\gamma_b = 75$
$x = 0$
$x = 0.25$
$x = 0.5$
$x = 0.75$
$x = 1$
c_a
c_b
A_b
A_a

Brief Report |
20 December 2022

Probable causal association between Australia's new regime of high all-cause mortality and its COVID-19 vaccine rollout

Denis G. Rancourt,[1,] * PhD ; Marine Baudin,[2] PhD ; Jérémie Mercier,[2] PhD

[1] Correlation Research in the Public Interest (correlation-canada.org)

* denis.rancourt@alumni.utoronto.ca

[2] Santé Liberté OÜ (jeremie-mercier.com)

This report is simultaneously posted on several websites, including:

https://correlation-canada.org/

https://denisrancourt.ca/

https://www.researchgate.net/profile/Marine-Baudin

https://ocla.ca/

https://www.jeremie-mercier.com/

https://archive.today/

ABSTRACT: All-cause mortality by week in Australia shows that there was no detectable excess mortality 13 months into the declared pandemic, followed by a step-wise increase in mortality in mid-April 2021, synchronous with the rollout of the COVID-19 vaccine prioritizing elderly, disabled and aboriginal residents. The excess mortality in the vaccination period (mid-April 2021 through August 2022; 14 % larger all-cause mortality than in recent pre-vaccination periods of same time duration; 62 million administered vaccine doses) was 31±1 thousand deaths, which is more than twice the deaths registered as from or with COVID-19. In addition, a sharp peak in all-cause mortality (mid-January to mid-February 2022; 2,600 deaths) is synchronous with the rapid rollout of the booster (9.4 million booster doses, same time period), and is not due to a climatic heatwave. We give thirteen numbered arguments as to why we conclude that the excess mortality in Australia is causally associated with the COVID-19 vaccine. The corresponding vaccine injection fatality ratio (vIFR) is approximately 0.05 %, which we compare to estimated vIFR values from the USA Vaccine Adverse Event Reporting System (VAERS) and from all-cause mortality data for India, Southern states of the USA, Michigan (USA) and Ontario (Canada).

Australia experienced a significant and sustained increase in all-cause mortality, starting with its COVID-19 vaccine rollout aimed at high-risk residents in mid-April 2021, whereas it saw no detectable excess all-cause mortality up to that point during 13 months of a pandemic that was declared by the World Health Organization (WHO) on 11 March 2020.

Starting in mid-April 2021, the all-cause mortality per week in Australia shows a sustained increase of >10 %, during which it never returns to its seasonal low value (of approximately 3,000 deaths/week) and attains highs of >4,000 deaths/week in June-

July-August 2022. The step-wise increase in all-cause mortality remains large up to the final date of presently consolidated official government statistics (week-34 of 2022, week ending 28 August 2022) (Australian Bureau of Statistics, 2022a).

Over the measured period of the step-wise increase in all-cause mortality (mid-April 2021 through August 2022; 14 % larger all-cause mortality than in recent pre-vaccination periods of same time duration; 62 million administered vaccine doses) there are 31±1 thousand excess deaths of all causes in Australia, whereas no excess deaths are detected in the prior 13-month period since a pandemic was declared (mid-March 2020 through mid-April 2021).

The excess all-cause mortality following the COVID-19 vaccine rollout (31,000 deaths, mid-April 2021 through August 2022) is more than twice the total number of deaths registered as being from or with COVID-19 (14,014 deaths, 1 January 2020 through 29 August 2022; WHO, consulted 20 December 2022, https://covid19.who.int/region/wpro/country/au).

The above points are corroborated and illustrated in the following figures.

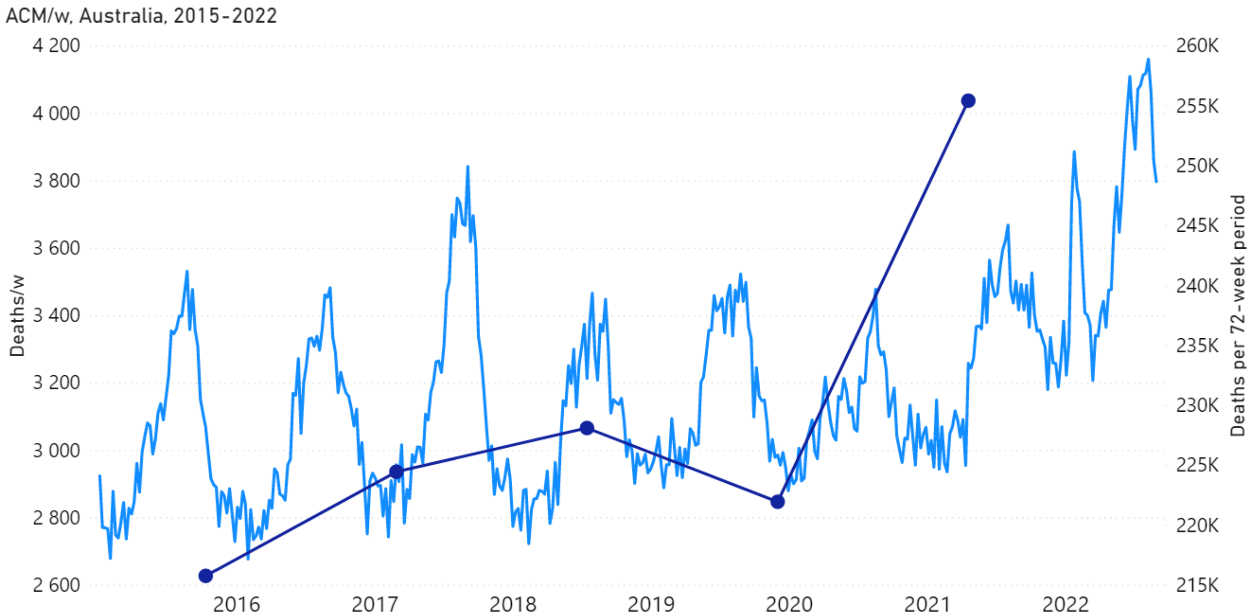


Figure 1A: All-cause mortality in Australia, all ages, from week-1 2015 (week ending 4 January 2015) through week-34 2022 (week ending 28 August 2022). Light-blue: All-cause mortality by week, left y-scale. Dark-blue: Integrated all-cause mortality over successive and non-overlapping 72-week periods (week-15 2021 through week-34 2022, for most recent period), right y-scale. Each point is positioned on the x-axis at the 1st week of its 72-week integration period. (Data source: Australian Bureau of Statistics, 2022a.)

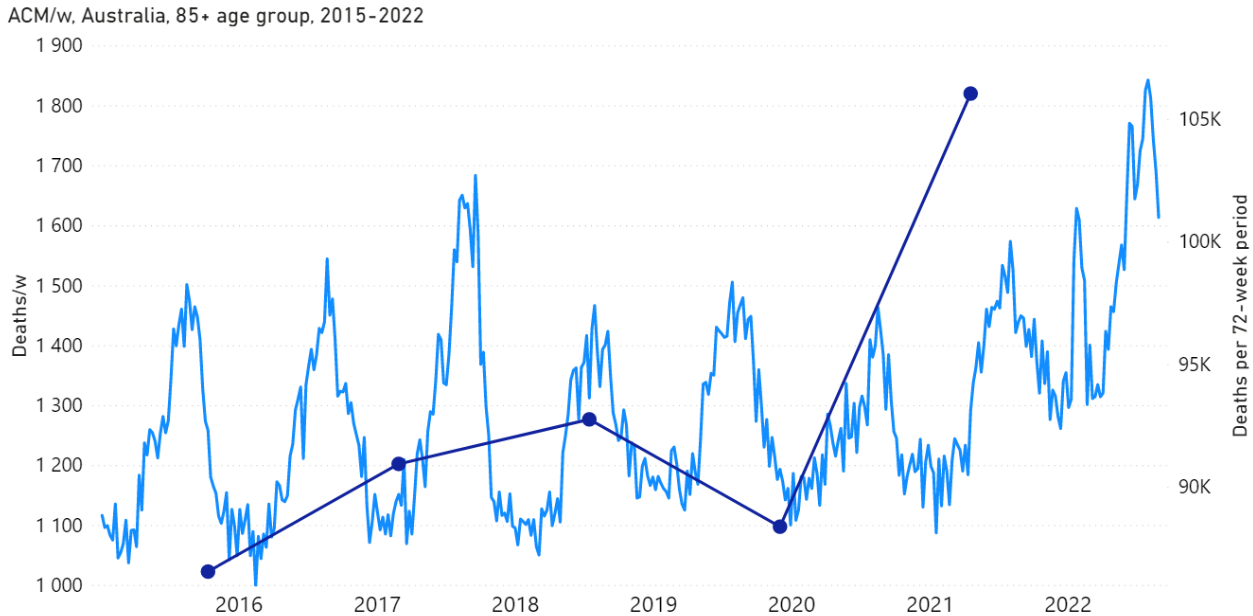


Figure 1B: All-cause mortality in Australia, ages 85+ years, from week-1 2015 (week ending 4 January 2015) through week-34 2022 (week ending 28 August 2022). Light-blue: All-cause mortality by week, left y-scale. Dark-blue: Integrated all-cause mortality over successive and non-overlapping 72-week periods (week-15 2021 through week-34 2022, for most recent period), right y-scale. Each point is positioned on the x-axis at the 1st week of its 72-week integration period. (Data source: Australian Bureau of Statistics, 2022a.)

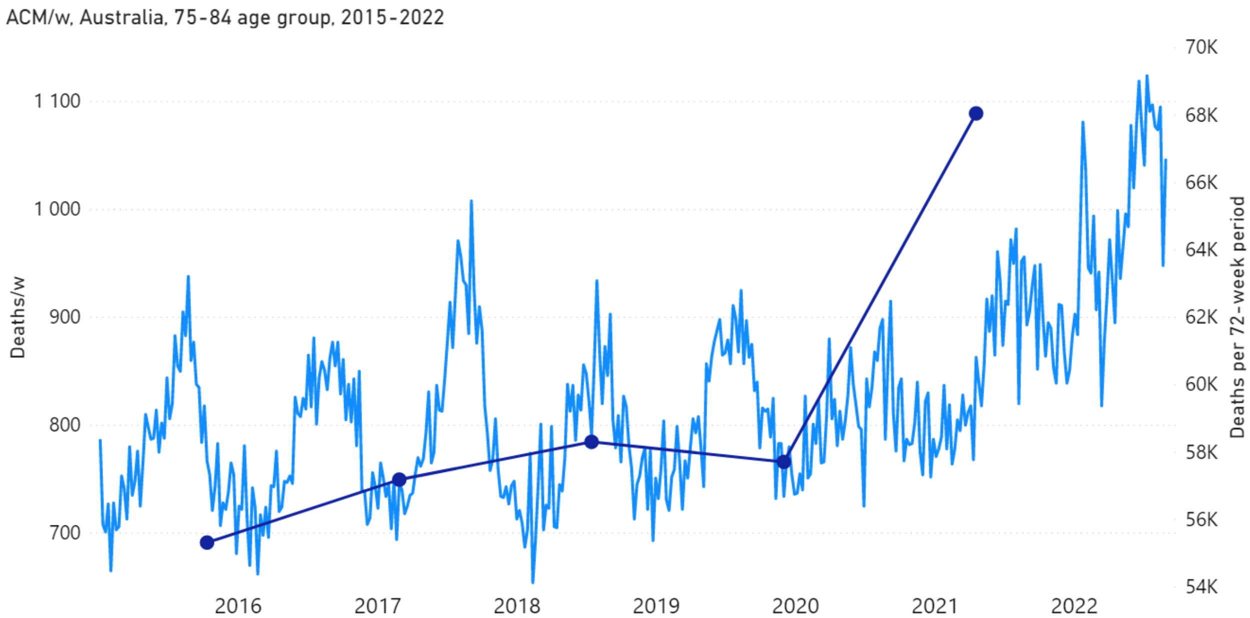


Figure 1C: All-cause mortality in Australia, ages 75-84 years, from week-1 2015 (week ending 4 January 2015) through week-34 2022 (week ending 28 August 2022). Light-blue: All-cause mortality by week, left y-scale. Dark-blue: Integrated all-cause mortality over successive and non-overlapping 72-week periods (week-15 2021 through week-34 2022, for most recent period), right y-scale. Each point is positioned on the x-axis at the 1st week of its 72-week integration period. (Data source: Australian Bureau of Statistics, 2022a.)

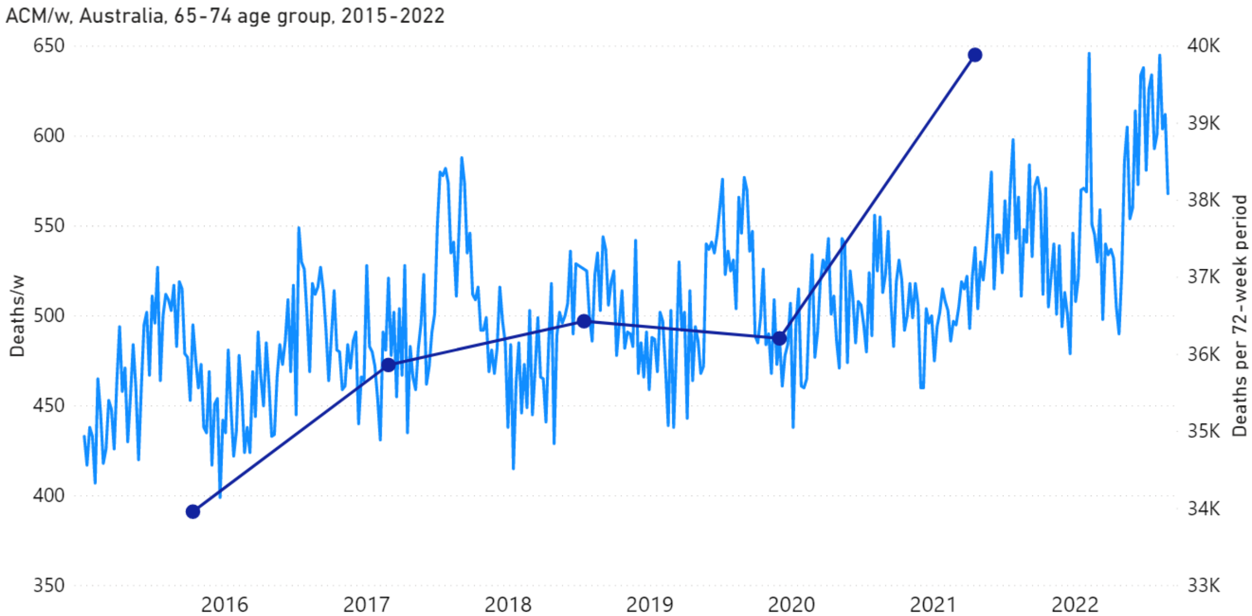


Figure 1D: All-cause mortality in Australia, ages 65-74 years, from week-1 2015 (week ending 4 January 2015) through week-34 2022 (week ending 28 August 2022). Light-blue: All-cause mortality by week, left y-scale. Dark-blue: Integrated all-cause mortality over successive and non-overlapping 72-week periods (week-15 2021 through week-34 2022, for most recent period), right y-scale. Each point is positioned on the x-axis at the 1st week of its 72-week integration period. (Data source: Australian Bureau of Statistics, 2022a.)

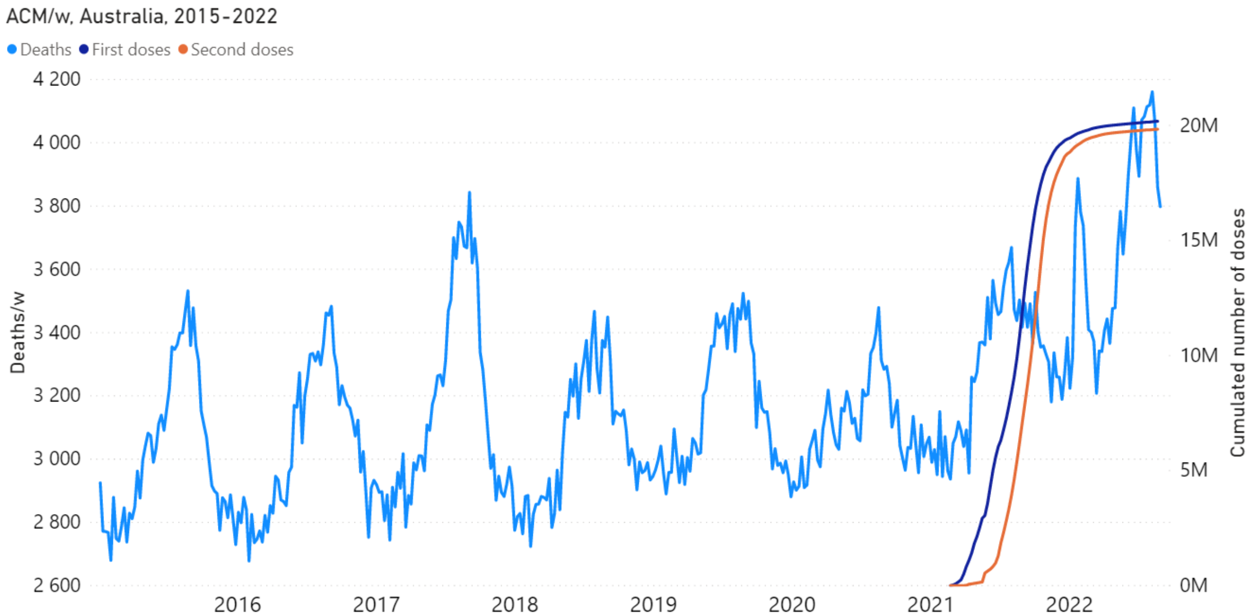


Figure 2: All-cause mortality in Australia, all ages, from week-1 2015 (week ending 4 January 2015) through week-34 2022 (week ending 28 August 2022), compared to the COVID-19 vaccine rollout. Light-blue: All-cause mortality by week, left y-scale. Dark-blue: Cumulative 1st doses of the vaccine. Orange: Cumulative 2nd doses of the vaccine. (Data sources: Australian Bureau of Statistics (2022a); and https://www.covid19data.com.au/vaccines, consulted on 14 December 2022.)

The vaccine rollout is shown in more detail as follows.

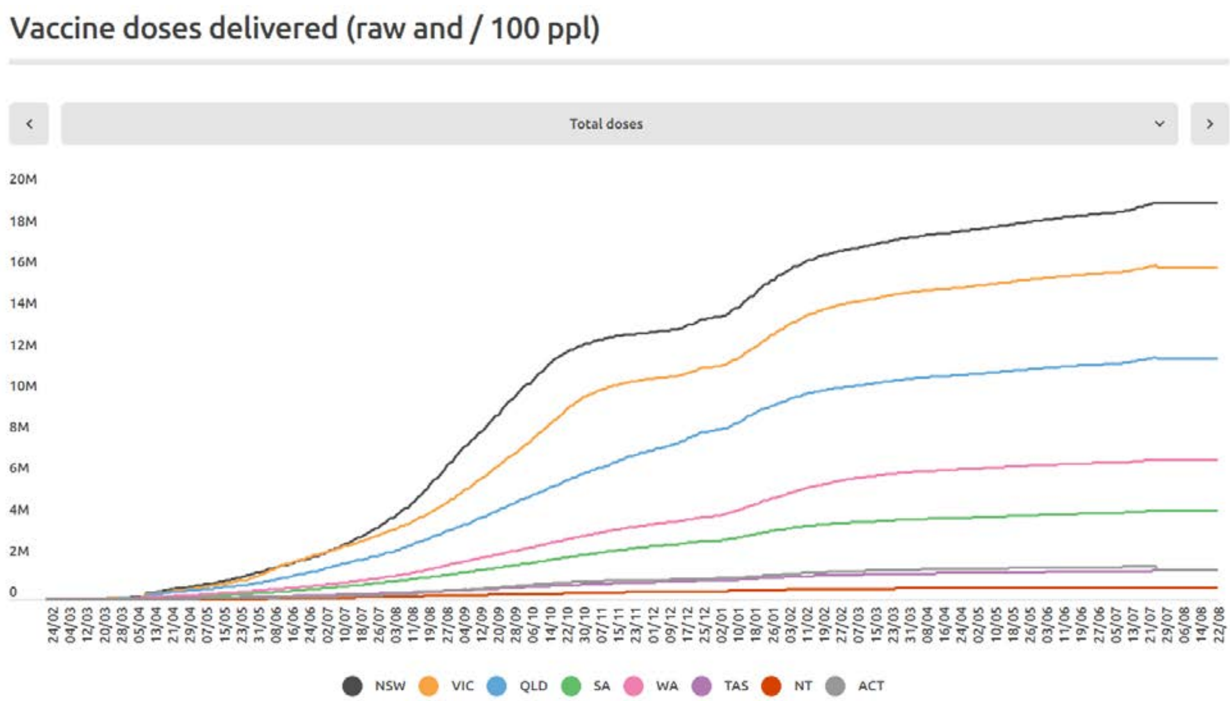


Figure 3A: Cumulative COVID-19 vaccine doses administered (all dose types) by time (24 February 2021 through 22 August 2022) by state in Australia (as indicated, in the sequence NSW, VIC, QLD, SA, WA, TAS, NT, ACT). (Source: https://www.covid19data.com.au/vaccines, accessed 20 December 2022.)

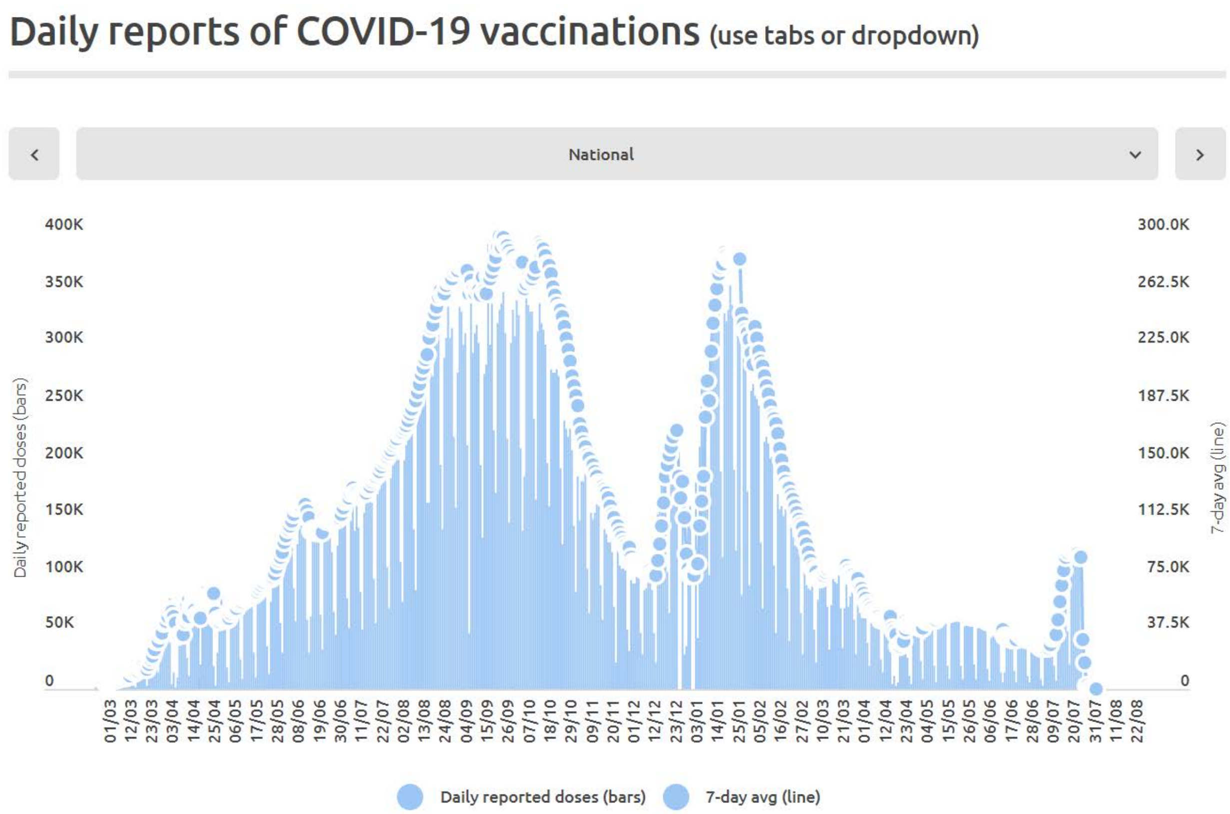


Figure 3B: Daily and 7-day average daily reported COVID-19 vaccine doses (all dose types) administered by time (1 March 2021 through 22 August 2022) in Australia. (Source: https://www.covid19data.com.au/vaccines, accessed 20 December 2022.)

Mortality and vaccination data specifically for the state of Victoria (VIC), Australia, is shown, for example, as follows.

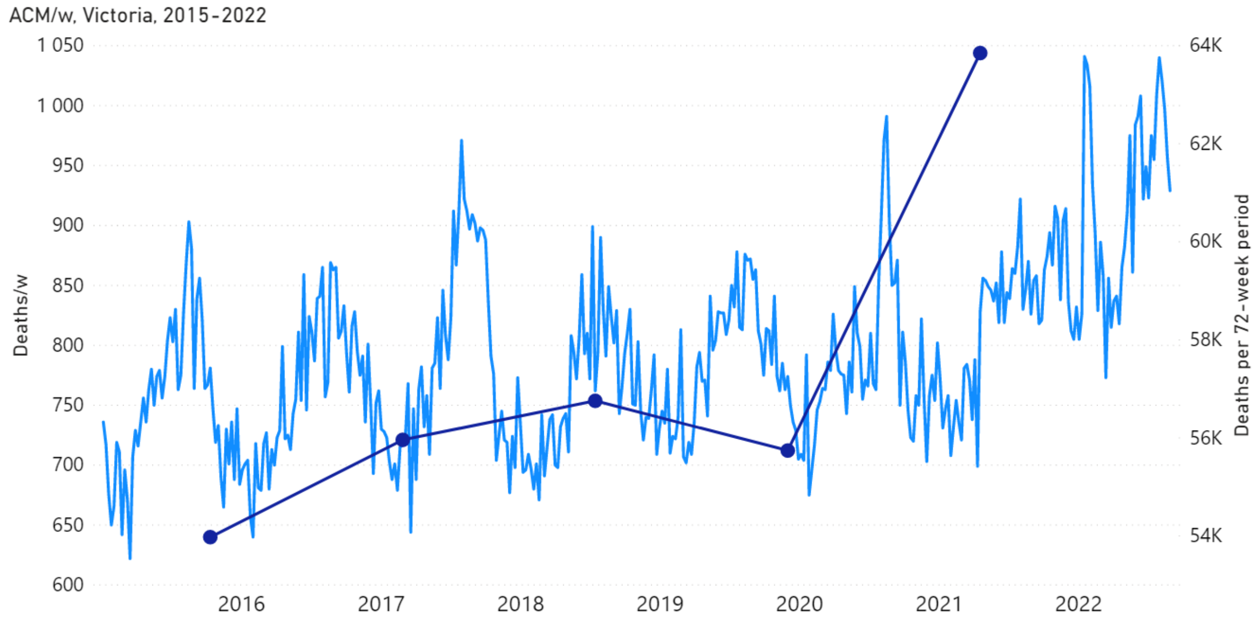


Figure 4A: All-cause mortality in the state of Victoria (VIC), Australia, all ages, from week-1 2015 (week ending 4 January 2015) through week-34 2022 (week ending 28 August 2022). Light-blue: All-cause mortality by week, left y-scale. Dark-blue: Integrated all-cause mortality over successive and non-overlapping 72-week periods (week-15 2021 through week-34 2022, for most recent period), right y-scale. Each point is positioned on the x-axis at the 1st week of its 72-week integration period. (Data source: Australian Bureau of Statistics, 2022a.)

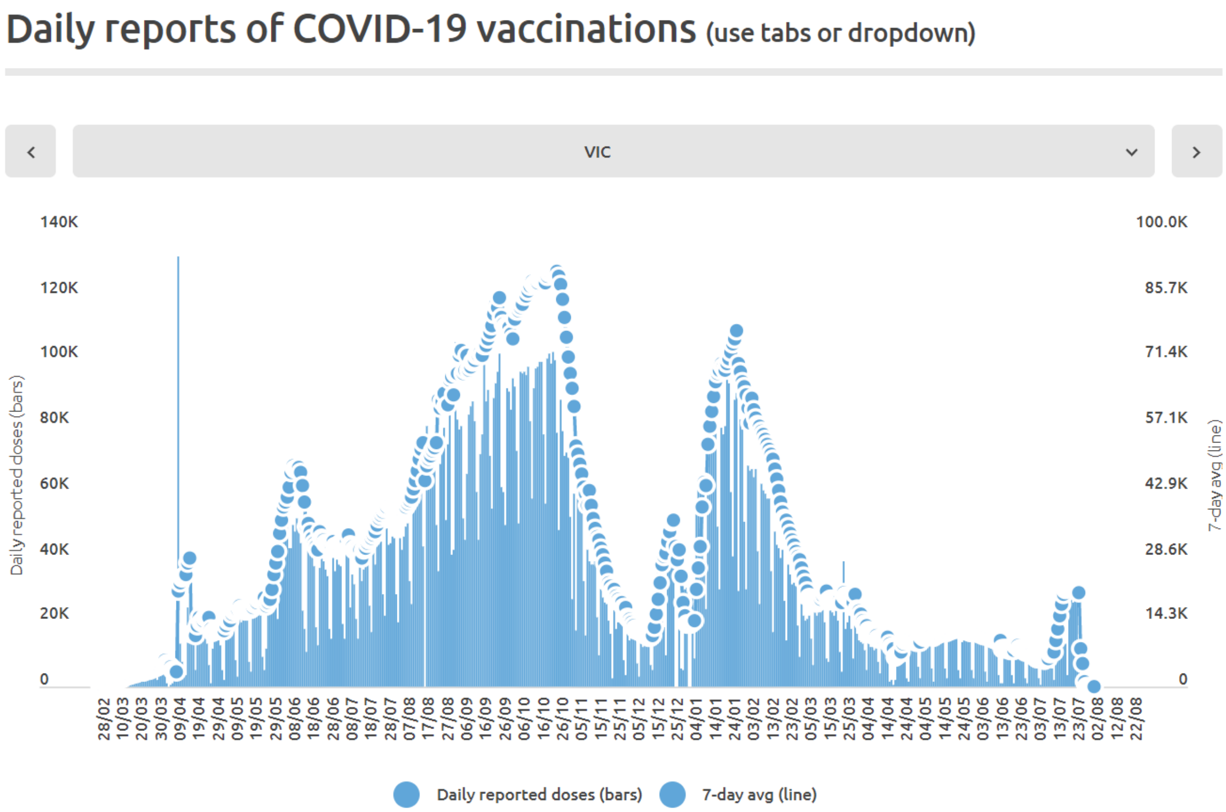


Figure 4B: Daily and 7-day average daily reported COVID-19 vaccine doses (all dose types) administered by time (28 February 2021 through 22 August 2022) in the state of Victoria (VIC), Australia. (Source: https://www.covid19data.com.au/vaccines, accessed 20 December 2022.)

The step-wise increase in mortality is evident in Figure 1 (A through C), and it is synchronous with the COVID-19 vaccine rollout (Figures 2, 3 and 4).

The step-wise transition to a regime of larger all-cause mortality is also seen in the different states of Australia. The example of Victoria is shown in Figure 4. The same phenomenon occurs in the all-cause mortality of all the eight states of Australia, although not clearly in NT (Northern Territory) (Appendix 1).

In addition to the above-described step-wise change in regime of all-cause mortality, there is a prominent peak in all-cause mortality, having a full duration of seven weeks, from mid-January to mid-February 2022. It is not consistent with a seasonal feature and it is synchronous with a large burst in COVID-19 vaccine dose delivery (Figures 1, 3B

and 4), which was the rollout of the booster (3rd doses) in Australia. The said 7-week-duration peak in all-cause mortality is prominent in the states NSW, QLD and VIC, but is essentially not present in the other states (Appendix 1). The booster rollout is shown in the following Figures 5 and 6.

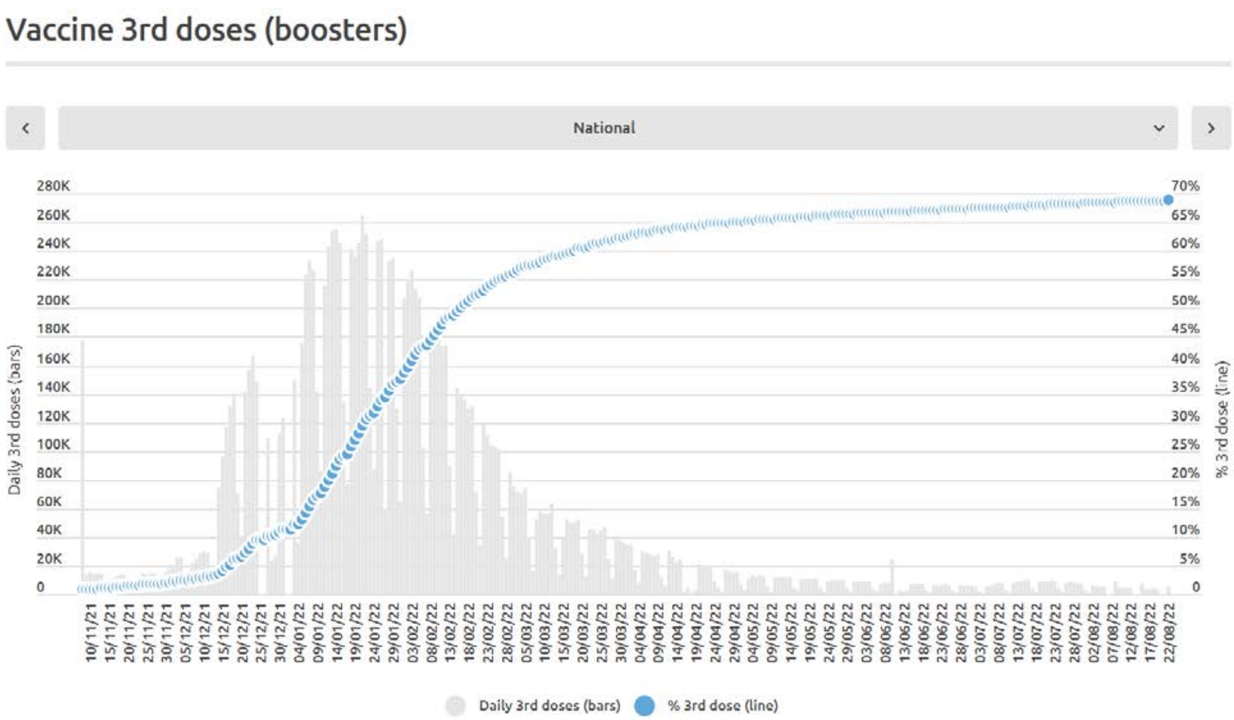


Figure 5: Daily and cumulative booster (3rd doses) rollout in Australia. The time axis is from 10 November 2021 through 22 August 2022. (Source: https://www.covid19data.com.au/vaccines, accessed 20 December 2022.)

Direct comparisons between all-cause mortality by week for the mid-January to mid-February 2022 peak and booster delivery by week are shown below, for Australia and for the states NSW, VIC and QLD (Figures 6A through 6D).

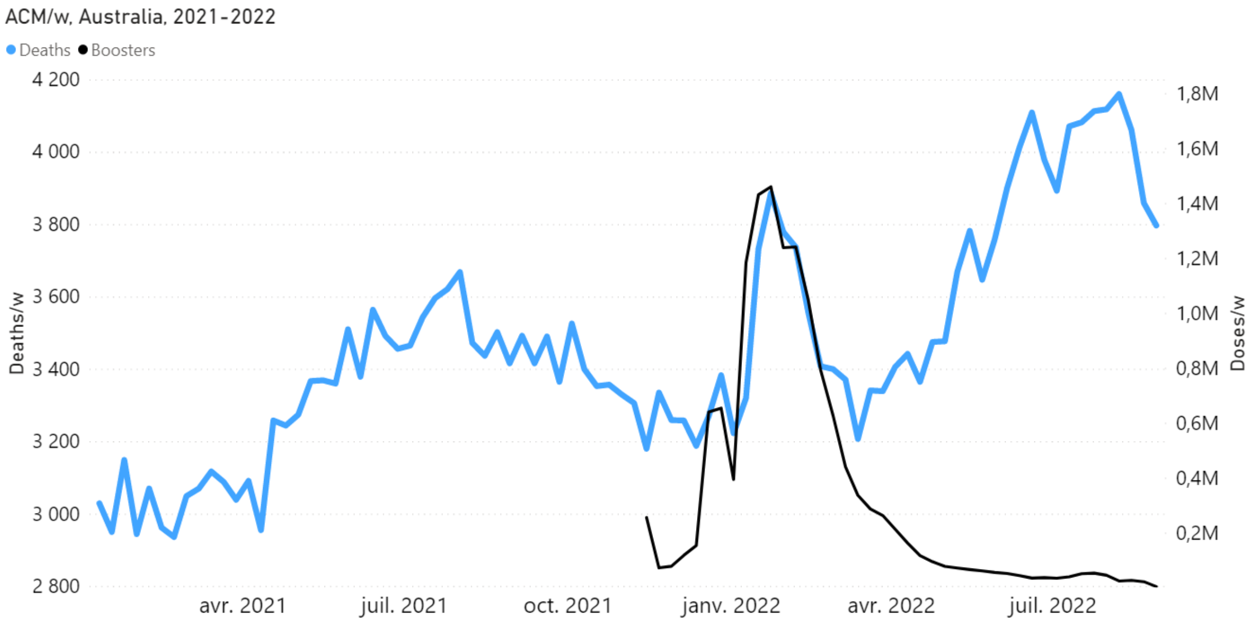


Figure 6A: Highlight of the mid-January to mid-February 2022 mortality peak, in relation to booster (3rd doses) delivery, in Australia. All-cause mortality by week (light-blue) and booster doses delivered by week (black) from 2021 to 2022. (Data sources: Australian Bureau of Statistics (2022a); and https://www.covid19data.com.au/vaccines, consulted on 14 December 2022.)

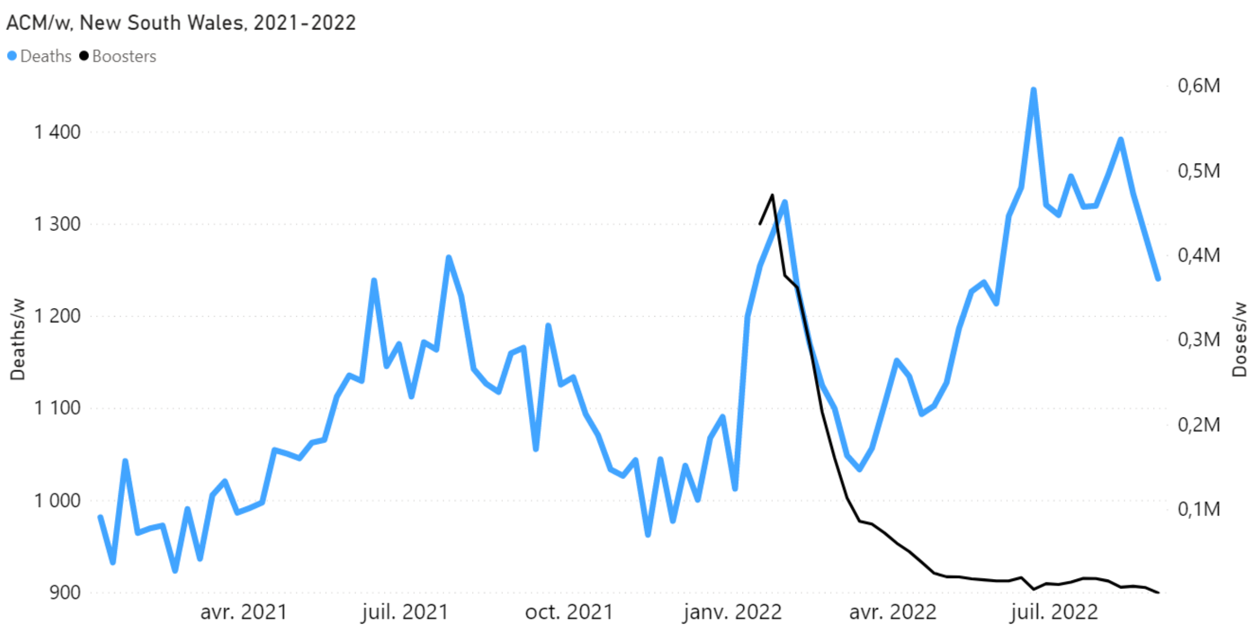


Figure 6B: Highlight of the mid-January to mid-February 2022 mortality peak, in relation to booster (3rd doses) delivery, in NSW (Australia). All-cause mortality by week (light-blue) and booster doses delivered by week (black) from 2021 to 2022. Both mortality and booster delivery are for NSW. (Data sources: Australian Bureau of Statistics (2022a); and https://www.covid19data.com.au/vaccines, consulted on 14 December 2022.)

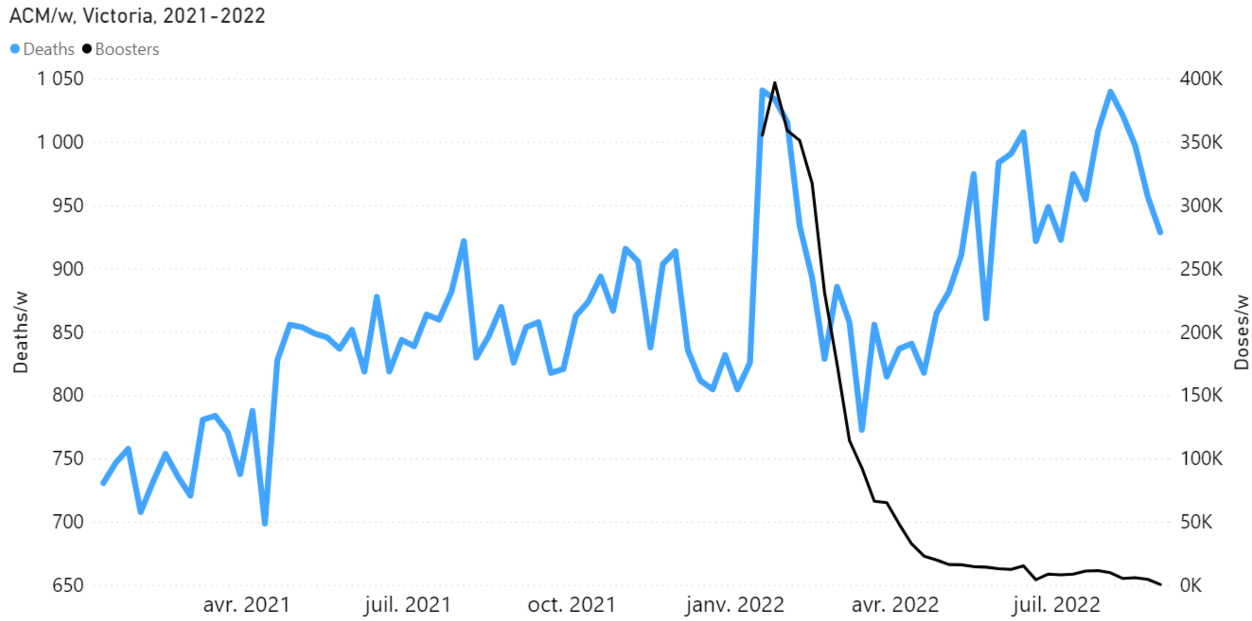


Figure 6C: Highlight of the mid-January to mid-February 2022 mortality peak, in relation to booster (3rd doses) delivery, in VIC (Australia). All-cause mortality by week (light-blue) and booster doses delivered by week (black) from 2021 to 2022. Both mortality and booster delivery are for VIC. (Data sources: Australian Bureau of Statistics (2022a); and https://www.covid19data.com.au/vaccines, consulted on 14 December 2022.)

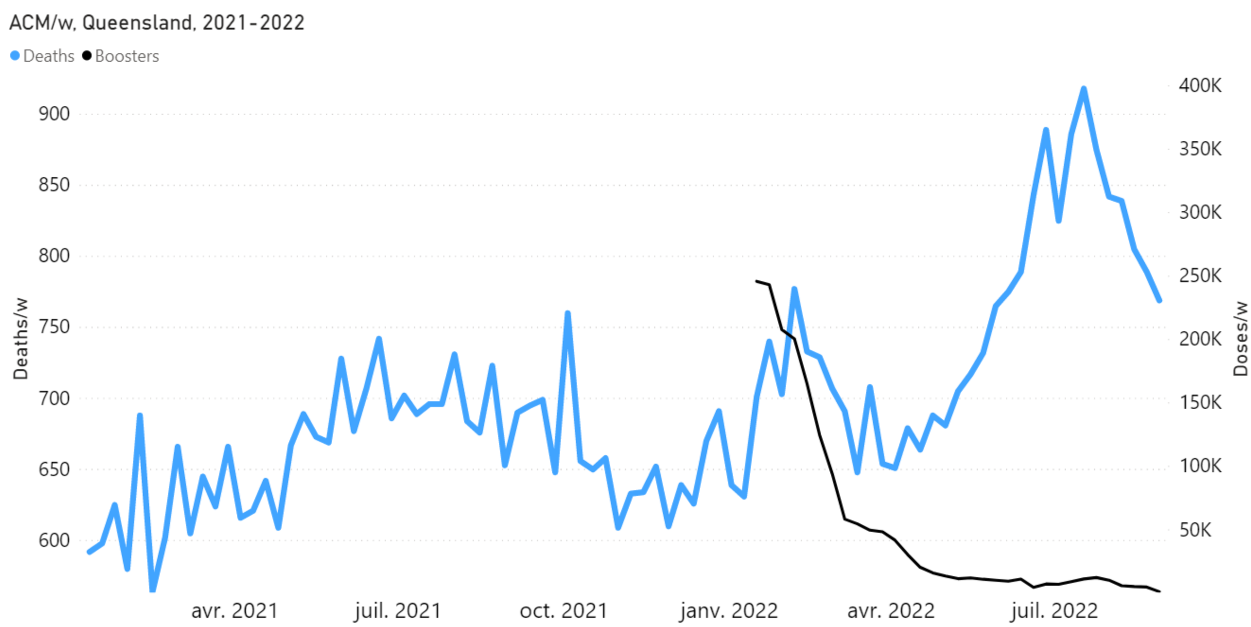


Figure 6D: Highlight of the mid-January to mid-February 2022 mortality peak, in relation to booster (3rd doses) delivery, in QLD (Australia). All-cause mortality by week (light-blue) and booster doses delivered by week (black) from 2021 to 2022. Both mortality and booster delivery are for QLD. (Data sources: Australian Bureau of Statistics (2022a); and https://www.covid19data.com.au/vaccines, consulted on 14 December 2022.)

The integrated excess mortality in the 7-week-duration peak, relative to its baseline, is approximately 2,600 deaths, compared to approximately 9.4 million booster doses delivered over the duration of the mortality peak. This corresponds to a vaccine injection fatality ratio (vIFR) of approximately 0.03 %, which in turn is not too different from the vIFR of 0.008 % for 65+ year old USA subjects injected with the Janssen vaccine, calculated from the Vaccine Adverse Event Reporting System (VAERS) data by Hickey and Rancourt (2022) (their Table 1).

An alternative hypothesis for the 7-week-duration mortality peak would be that it was caused by an Australian summer heatwave affecting Eastern Australia. This hypothesis is not tenable with the climatic and mortality data, which we demonstrate in Appendix 2.

For the following reasons (presented as numbered points), taken together, we conclude that the 16-month (mid-April 2021 through August 2022) sustained regime of large excess all-cause mortality in Australia may largely or predominantly be caused by its vaccine rollout, including the booster (3rd doses).

1 - There is a clear temporal association between the new regime of heightened all-cause mortality and the vaccine rollout, whereas Australia did not have detectable excess mortality up to the start of the rollout, during 13 months of a pandemic that was declared by the WHO on 11 March 2020. (Figures 1, 2, 4 and 6; and Appendix 1)

2 - The excess mortality in the vaccination period (mid-April 2021 through August 2022) for Australia (all ages) is 31,000 (±1,000) deaths (Figure 1A), which is more than twice the total number of deaths registered as being from or with COVID-19 (14,014 deaths, 1 January 2020 through week ending 29 August 2022; WHO, consulted 15 December 2022, https://covid19.who.int/region/wpro/country/au).

Note that the percentage of total COVID-19-assigned deaths that are "with COVID-19" (rather than "from COVID-19") varies between approximately 10 % and 30 %, in the period January 2022 through August 2022 (Australian Bureau of Statistics, 2022b; their figure entitled "Proportion of deaths from and with COVID-19 during the Omicron wave", and see "Proportion of deaths from and with COVID-19 during the Omicron wave by state of registration"). Here, death "from COVID-19" means that COVID-19 is assigned as "the underlying cause of death as the disease or condition that initiated the train of morbid events leading to death", whereas other diseases and conditions reported as contributing to death are "referred to as associated causes" (Australian Bureau of Statistics, 2022c). In fact, 95.4 % of deaths "from COVID-19" in Australian death certificates had non-COVID-19 "causal sequences of events" and/or "pre-existing chronic conditions" (Australian Bureau of Statistics, 2022c; their table entitled "Number of deaths due to COVID-19 that has associated conditions").

The question is unavoidable: Why would Australians suddenly (at the start of the vaccine rollout) start dying in excess of something mostly if not entirely other than COVID-19, after 13 months of a declared pandemic during which there was no detectable excess all-cause mortality?

3 - The mean vIFR in the vaccination period (mid-April 2021 through August 2022) for Australia, therefore, would be:

31 K deaths / 62 M vaccine doses[1] = 0.05 %

which is larger than the vIFR of 0.008 % for 65+ year old USA subjects injected with the Janssen vaccine, calculated from the VAERS data (Hickey and Rancourt, 2022; their Table 1), and smaller than the estimated 1 % calculated for the excess mortality event in India (Rancourt, 2022), and for excess mortality peaks for several Southern states of

[1] Cumulative COVID-19 vaccine doses administered: All doses, including boosters, are counted individually; administered 14 April 2021 through 25 August 2022, 63.01M - 1.36M = 62M. Our World in Data, accessed 16 December 2022: https://ourworldindata.org/explorers/coronavirus-data-explorer?facet=none&Interval=Cumulative&Relative+to+Population=false&Color+by+test+positivity=false&country=~AUS&Metric=Vaccine+doses

the USA (Rancourt et al., 2022). As such, the 0.05 % estimated mean vIFR for Australia is within an expected range for real-world circumstances.

4 - In addition to the above-described vaccination-period regime of all-cause mortality (mid-April 2021 through August 2022), there is a prominent peak in all-cause mortality from mid-January to mid-February 2022, having a full duration of seven weeks, which is synchronous with a large burst in COVID-19 vaccine dose delivery (Figures 1, 3B, 4 and 6). The said large burst in vaccine dose delivery was the rollout of the booster (3rd doses) in Australia (Figures 5 and 6).

We stress that Figure 6, showing a high degree of synchronicity (in both position and width) between the mid-January to mid-February 2022 all-cause mortality peak and the booster (3rd doses) delivery pattern, with the booster delivery surge generally leading the mortality surge by approximately 1 week, represents strong evidence for a causal relation; the strongest we have seen in all-cause mortality data.

5 - The said prominent peak in all-cause mortality from mid-January to mid-February 2022 has an integrated excess mortality in its 7-week duration, relative to its baseline, of approximately 2,600 deaths, compared to approximately 9.4 million booster doses delivered over the duration of the mortality peak. This corresponds to a calculated vIFR for the specific mortality peak:

2.6 K deaths / 9.4 M vaccine doses[2] = 0.03 %

which is comparable in value to that obtained (0.05 %) for the mean vIFR in the vaccination period (mid-April 2021 through August 2022) for Australia.

[2] Estimated using cumulative COVID-19 vaccine doses administered: All doses, including boosters, are counted individually; administered 8 January 2022 through 21 February 2022, 53.4M - 44.0M = 9.4M. Our World in Data, accessed 16 December 2022: https://ourworldindata.org/explorers/coronavirus-data-explorer?facet=none&Interval=Cumulative&Relative+to+Population=false&Color+by+test+positivity=false&country=~AUS&Metric=Vaccine+doses

6 - The impact of the rollout would be sudden, as observed (Figures 1, 2, 4A and 6; and Appendix 1), because Australia prioritized elderly, disabled and aboriginal residents (Australian Government - Department of Health and Aged Care, 2021).

7 - The step-wise increase in all-cause mortality, into the regime of excess all-cause mortality (mid-April 2021 through August 2022) occurs simultaneously in mid-April 2021 across all of Australia, in the eight states (see Appendix 1), rather than showing any distribution of starting times, which would be compatible with a spreading infectious disease seeding different regions at different times and spreading at different rates depending on regional differences of social and health conditions.

In this regard, theoretical models of spreading and emerging pandemics show high sensitivity of dynamic outcomes to seeding, societal population size, and inferred social and health conditions (Parham and Michael, 2011; Hasegawa and Nemoto, 2016; Ma et al., 2022).

8 - The VAERS data of the USA unambiguously shows excess all-cause deaths immediately following injections with each of the three types of COVID-19 vaccines used in the USA, with a prominent peak within 5 days of injection and an exponentially decaying excess mortality extending 2 months following injection (Hickey and Rancourt, 2022; see their Figs. S3 through S5). The integrated mortality by number of injections following injection (injection toxicity or vIFR) increases exponentially with age, as does the batch to batch variability of toxic effect (Hickey and Rancourt, 2022; see their Fig. S6). The latter observations of exponential increases with age mean that the injections represent fatal challenges in proportion to frailty of the subject.

9 - Detailed histopathological and immunohistochemical autopsy studies have demonstrated that the COVID-19 vaccines are causes of death, both in otherwise healthy subjects and in elderly subjects with comorbidities (Choi et al., 2021; Schneider et al., 2021; Sessa et al., 2021; Gill et al., 2022; Mörz, 2022; Schwab et al., 2022; Yoshimura et al., 2022).

10 - The Australian Government interprets both test results (cases) and the mortality as occurring in four "waves", which it describes by time period as follows (Australian Bureau of Statistics, 2022b):

- "Wave 1: as occurring between March and May 2020. The predominant variant during Wave 1 was the original virus strain.
- Wave 2: as occurring between June and November 2020. Wave 2 predominantly occurred in Victoria. The variant during Wave 2 was the original virus strain.
- Delta wave: as occurring between July and December 2021.
- Omicron wave: as occurring during 2022 (until the end of September 2022). Due to the length of this wave and the higher number of deaths […]."

We have not found any study establishing a scientific basis for the Australian Government's assignation of these waves. Furthermore, the said Government's assignation is irreconcilable with:

i. the absence of detected excess mortality in March-May 2020 (Figure 1; and Appendix 1),
ii. the absence of detected excess mortality in Australia (Figure 1A) and in Victoria (Figure 4A) in the period June-November 2020 (and see Appendix 1),
iii. a Delta-variant wave (July-December 2021) that would have missed both the mid-April 2021 step-wise surge in excess all-cause mortality and the 7-week-duration mid-January to mid-February 2022 peak in excess all-cause mortality, and
iv. an Omicron-variant wave (2022) that would have caused two distinct and prominent features in excess all-cause mortality, namely the mid-January to mid-February 2022 7-week-duration peak and the large surge that followed starting in May 2022 (Figure 1A).

The official interpretive situation is similar, although less sophisticated, to that employed by Dhar et al. (2021) who postulated that the April-July 2021 "second wave" event in Delhi (the capital city of India) was due to the Delta variant, which would have quickly

swept Delhi to become predominant because it would have higher transmissibility and larger immune escape than concomitantly circulating variants. However, Dhar et al. estimate the needed characteristics of Delta by fitting a model to the epidemiological data and to the variant predominance estimated by genomic measurements from small non-randomized cohorts. Leaving aside the large known and unknown uncertainties throughout their exercise, basically, the inferred characteristics of Delta are obtained by fitting to the data, rather than being independently measured in a controlled clinical trial. Under such circumstances, the mortality event creates an illusion of the needed Delta for Delhi, but an actual Delta cannot be concluded to have caused the mortality event.

Likewise, the Australian Government’s assignation of COVID-19 waves for Australia is merely a naming exercise of reported test results (case statistics), coupled to sparse and unreliable genomic measurements (Australian Government - Department of Health and Aged Care, 2022). The Australian Government’s assignation is contradicted by hard data of all-cause mortality by time.

11 - A similar synchronicity between vaccine dose delivery and excess all-cause mortality is observed in connection with the so-called “vaccine equity” campaigns in the USA. An anomalous fall-2021 peak was interpreted as being caused by the vaccines, and is prominent in the 25-64 years age group in 21 states of the USA, most notably including Alabama, Mississippi, Georgia, Florida and Louisiana (Rancourt et al., 2022). The data for Mississippi is shown below (Figure 7).

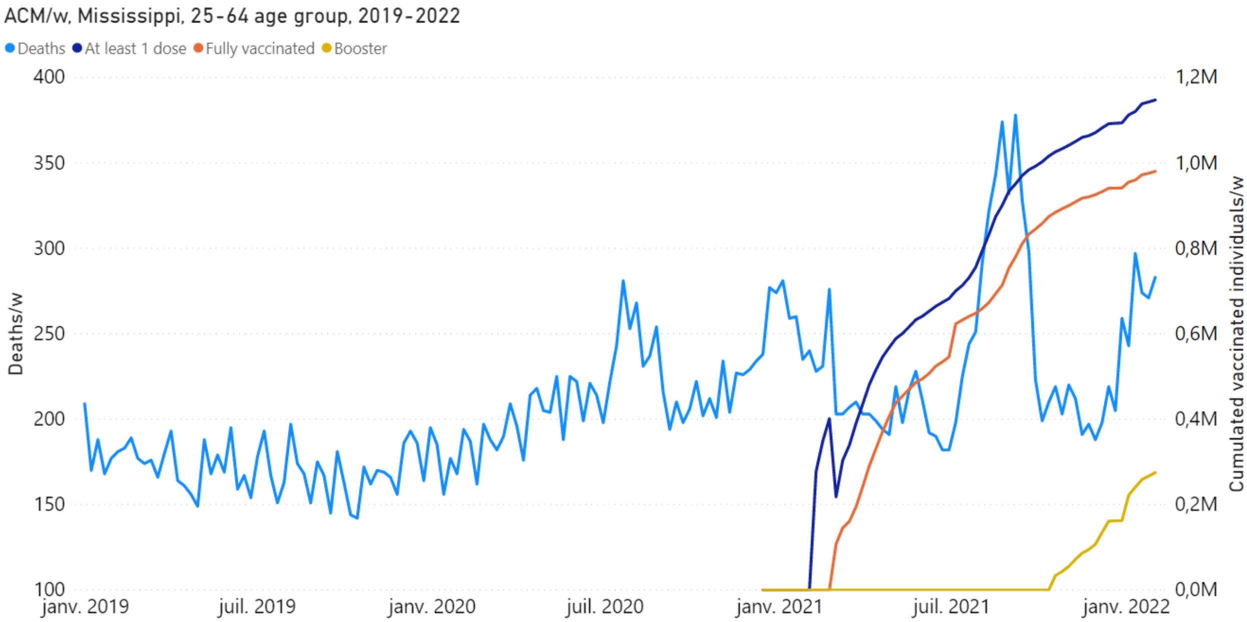


Figure 7: Rancourt et al. (2022), their Fig. 11B. All-cause mortality by week (light-blue), cumulated number of people with at least one dose of vaccine (dark-blue), cumulated number of fully vaccinated people (orange) and cumulated number of people with a booster dose (yellow) by week from 2019 to 2022, for 25-64 years age group in Mississippi. Data are displayed from week-1 of 2019 to week-5 of 2022.

In the study by Rancourt et al. (2022), it was concluded that significant (detectable by all-cause mortality) vaccine-induced mortality occurred primarily among fragile groups, characterized by high degrees of poverty, disability, obesity, diabetes, and high medication rates. The vaccine injection was seen as an additional challenge, often accelerating and causing death in residents with comorbidities.

12 - Another example of probably causal synchronicity between a rapid COVID-19 vaccine rollout prioritizing elderly, frail and disabled residents and large excess all-cause mortality is that of India (Rancourt, 2022). In that case, the early rollout of the vaccine in April-July 2021 was devastating, causing the deaths of approximately 3.7 million residents, on administering approximately 350 million doses of the vaccine (in a population of 1.39 billion). This corresponds to an effective vIFR (per-dose toxicity) of approximately 1 %. It is also approximately the same vIFR (1 %) as is consistent with the anomalous fall-2021 peak in excess all-cause mortality occurring in high-poverty

states of the USA, which was interpreted as being caused by the vaccine (Rancourt et al., 2022; and see the data for Mississippi shown in Figure 7).

Clearly, frail residents are susceptible to being fatally harmed by the injection and should be protected against state-run injection campaigns implemented without stringent individual clinical risk assessment. It appears that the population-wide COVID-19 vIFR can be as large as 1 % (India, Southern USA states), and is approximately 0.05 % in Australia.

Both India and Australia had virtually no detectable excess all-cause mortality after a pandemic was declared by the WHO, until their respective COVID-19 vaccine rollouts, which makes the synchronicity association relatively easy to assign.

13 - Two more examples of synchronicity between a rapid COVID-19 vaccine rollout prioritizing elderly and vulnerable residents and large excess all-cause mortality occur for Michigan, USA (Rancourt et al., 2022) and Ontario, Canada.

Key figures for Michigan, USA are as follows (Figure 8). The COVID-19 vIFR in the main rollout of the vaccine in Michigan is comparable in value to that for the vaccination period for Australia (0.05 %).

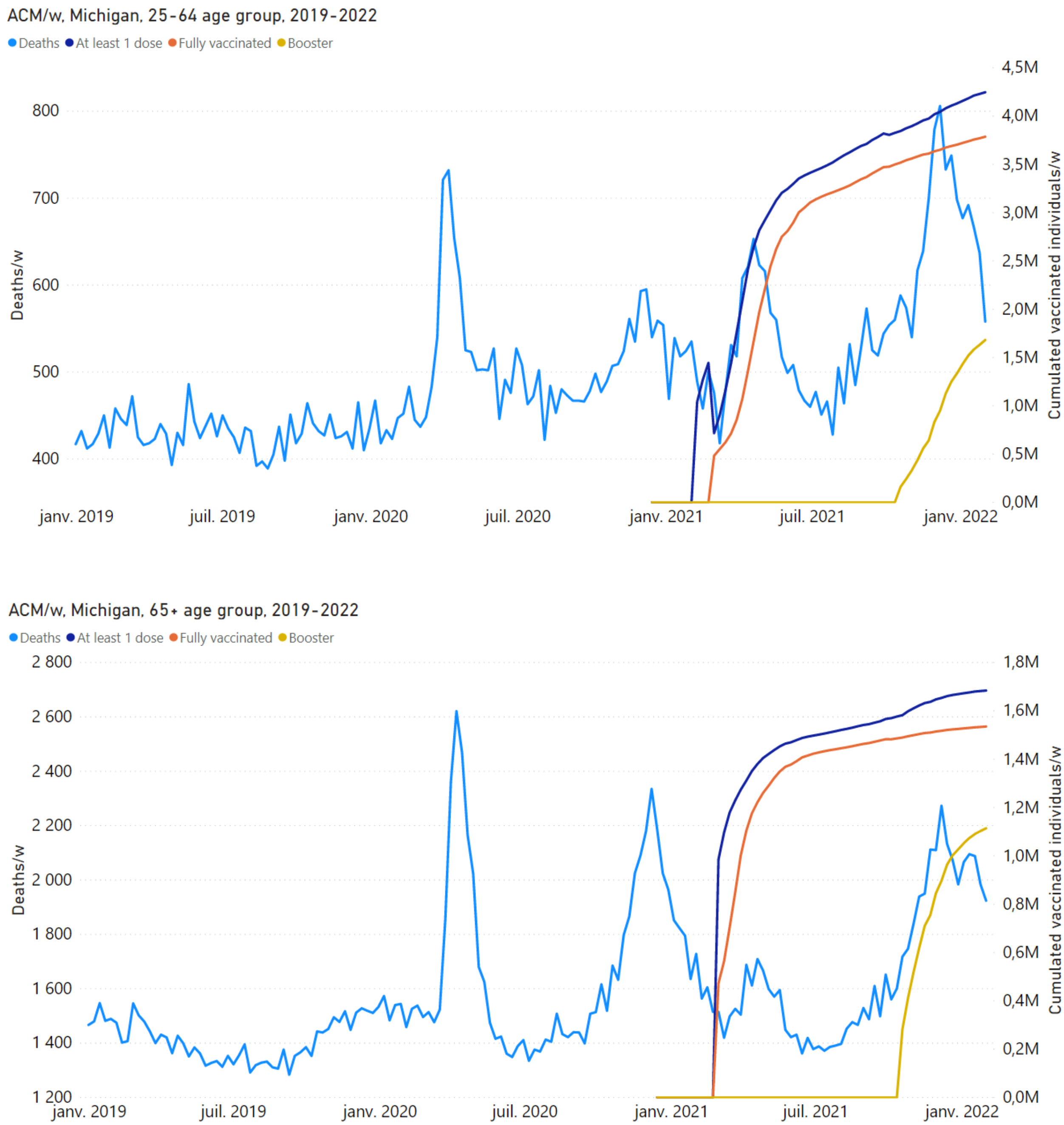


Figure 8: All-cause mortality by week (light-blue), cumulative number of people with at least one dose of vaccine (dark-blue), cumulative number of fully vaccinated people (orange) and cumulative number of people with a booster dose (yellow) by week from 2019 to 2022, and by age group for Michigan, USA. Data are displayed from week-1 of 2019 to week-5 of 2022. Upper panel: (Rancourt et al., 2022; their Figure 11G) Michigan, 25-64 years age group. For the 25-64 years age group, the vaccination data is for the 18-64 years age group. Lower panel: (Rancourt et al., 2022; their Figure 11H) Michigan, 65+ years age group. The discontinuous breaks in cumulative number of vaccinated individuals are artifacts.

A key figure for Ontario, Canada is as follows (Figure 9).

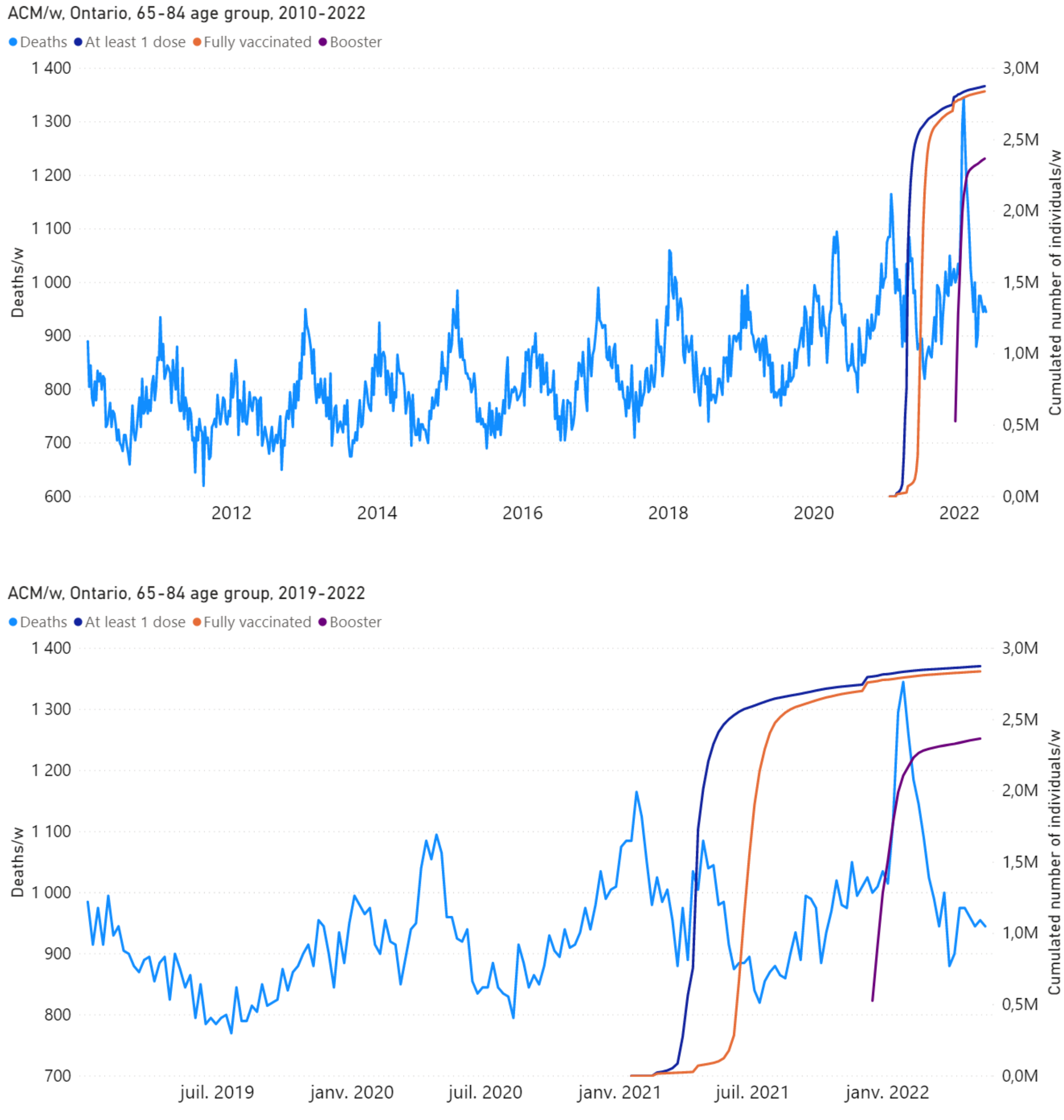


Figure 9: All-cause mortality by week (light-blue), cumulative number of people with at least one dose of vaccine (dark-blue), cumulative number of fully vaccinated people (orange) and cumulative number of people with a booster dose (purple) by week from 2010 to 2022 (upper panel), and from 2019 to 2022 (lower panel), in the province of Ontario, Canada. Both mortality and vaccination are for the age group 65-84 years. (Rancourt et al., manuscript in preparation)

A clear non-seasonal peak is seen in this age group (65-84 years) in Ontario, which is synchronous with the COVID-19 vaccine rollout to this age group (Figure 9); and a particularly large and sharp mortality peak is synchronous with the booster rollout to this age group the following winter season (Figure 9). Here, again, the corresponding COVID-19 vIFRs are comparable in value to that for the vaccination period for Australia (0.05 %).

As further discussion, we make the following observations and comments.

As outlined above, less than and approximately half of the excess deaths of all causes in the vaccination period are deaths registered as COVID-19 deaths. The COVID-19-registered deaths have the following properties (Australian Bureau of Statistics, 2022c):

i. Attribution of death "from COVID-19" versus "with COVID-19" is based on a qualitative evaluation susceptible to bias
ii. 95.4 % of deaths "from COVID-19" in Australian death certificates had non-COVID-19 "causal sequences of events" and/or "pre-existing chronic conditions"
iii. The deaths statistics by age and sex are typical of all-cause old-age deaths statistics in Western societies
iv. The three "most commonly certified acute disease outcomes of COVID-19" were: pneumonia (61.4 %), respiratory failure (15 %), and other infections (11.2 %)
v. The three most common pre-existing conditions in certified "with COVID-19" deaths were: chronic cardiac conditions (39.0 %), dementia (30.5 %), and chronic respiratory conditions (17.8 %)

Therefore, it is reasonable to infer that the vaccine injections caused death by providing an additional and significant challenge to already chronically frail or vulnerable subjects,

and that COVID-19 itself may not have provided a significant contribution, as we already demonstrated for the Southern states of the USA (Rancourt et al., 2022), and as is apparent for India (Rancourt, 2022).

In this context, and given the “most commonly certified acute disease outcomes of COVID-19”, it is important to note that Australia, like virtually all Western jurisdictions, dramatically reduced its antibiotic prescriptions after a pandemic was declared by the WHO (Gillies et al., 2021; Rancourt et al., 2022). This would mean that, not only were chronically frail residents challenged with the toxic injections, but they may also not have been provided the normal treatments against respiratory bacterial infections.

Finally, we note that there is starting to be some acknowledgement in the mainstream media suggesting that vaccine harm in Australia may be much larger than generally admitted by the medical establishment. The recent public testimony and submission to Parliament of former federal MP and former Australian Medical Association (AMA) president Dr. Kerryn Phelps stands out in this regard (Chung, 2022).

In conclusion, the declared pandemic would have had to entirely spare Australia any detectable deaths for more than a year, while it raged in many other places around the world, before it showed any virulence, suddenly in mid-April 2021, when vaccines coincidentally were being rolled out to the elderly and most vulnerable. In addition, a sharp peak in all-cause mortality (mid-January to mid-February 2022) would be synchronous with the rapid deployment of the vaccine booster (3rd doses) purely by coincidence, without any explanation (plausible or not) being provided.

On the contrary, our analysis leads us to conclude that the excess mortality in the vaccination period (31±1 thousand deaths, mid-April 2021 through August 2022; 14 % larger all-cause mortality than in recent pre-vaccination periods of same time duration; 62 million administered vaccine doses), which is more than twice the deaths registered as from or with COVID-19, and the sharp peak in all-cause mortality (mid-January to

mid-February 2022; 2,600 deaths), which is synchronous with the rapid rollout of the booster (9.4 million booster doses, same time period) are causally associated with the COVID-19 vaccine. We give thirteen numbered arguments as to why we make this conclusion.

The corresponding vaccine injection fatality ratio (vIFR) is approximately 0.05 %, which is intermediate between the value from VAERS for ages 65+ years with the Janssen vaccine in the USA (0.008 %) and the value for India's vaccine rollout and for Southern states of the USA subjected to "vaccine equity" campaigns (1 %).

Of course, this is diametrically opposite to the proposal that the COVID-19 vaccine would have saved any lives; a proposal that is not substantiated by extensive study of all-cause mortality data (Rancourt et al., 2022).

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APPENDIX 1:
Step-wise increase in all-cause mortality occurs in mid-April 2021 in all the states in Australia

Here, we show the all-cause mortality data for Australia and for each state of Australia (as labelled in the panels of Figure A1-F1), and including the 72-week vaccination period integrations, described in the present article.

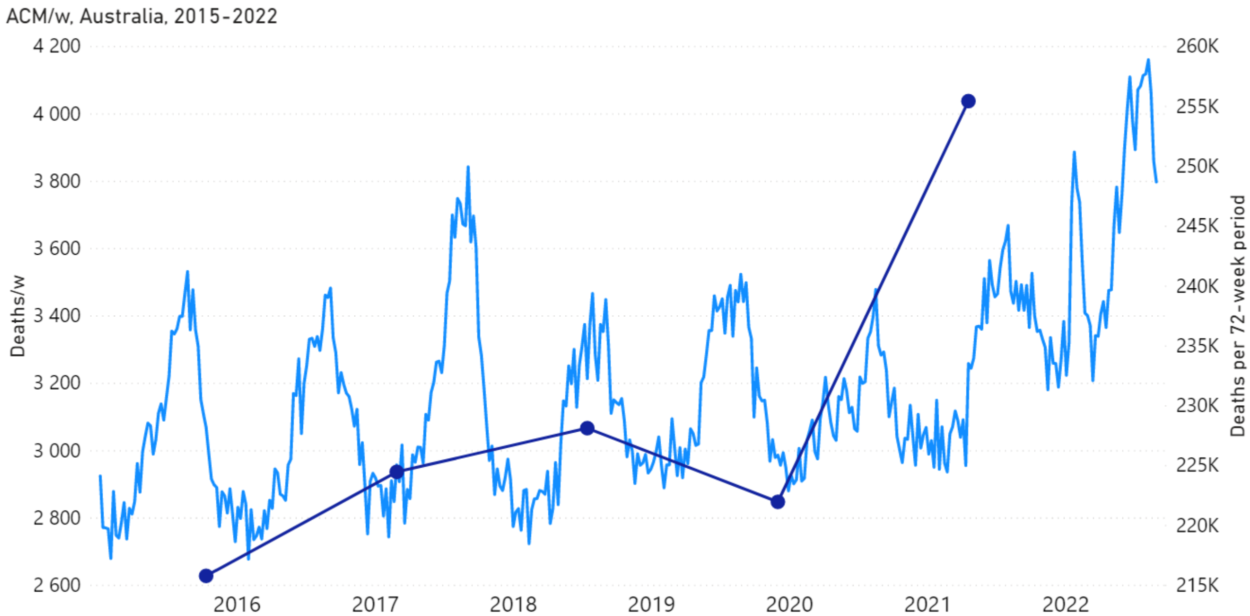
We also provide the following table of corresponding vaccine-period excess mortalities.

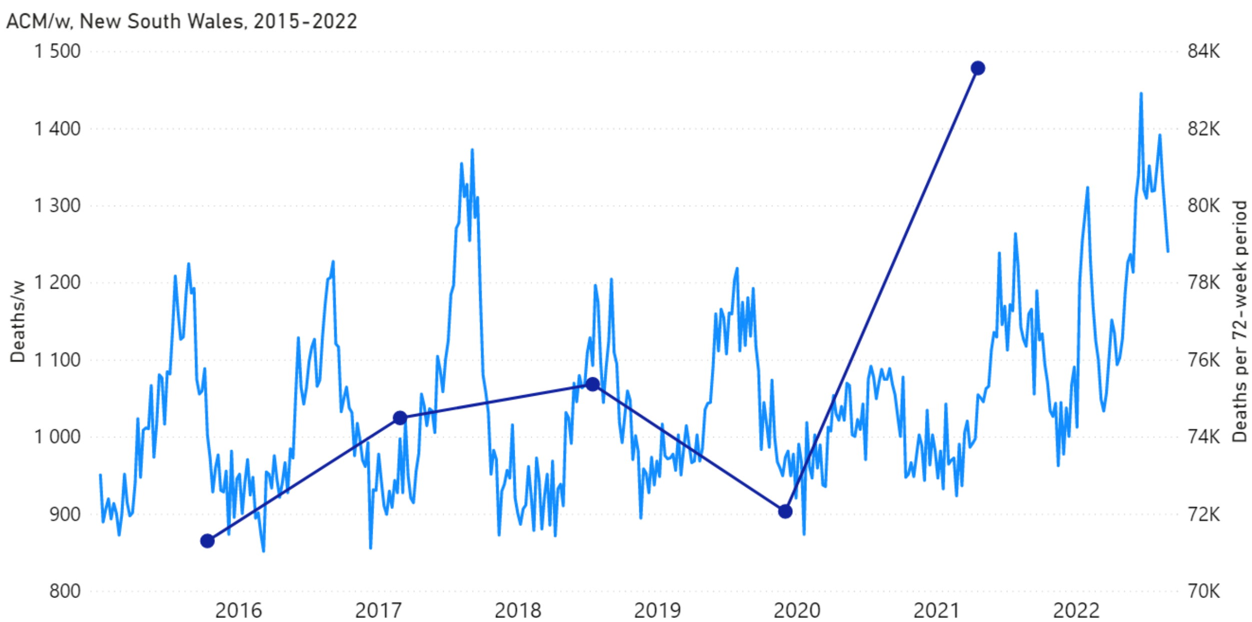
Table A1-T1: Integrated all-cause mortality (72 weeks), differences and ratios

State	Population (M) (2022)	Baseline Period (K)*	Vaccination Period (K)	Excess (K)	Excess /Baseline (%)	Excess deaths (per 100K)
Australia	25.979	224.5	255.5	31.0	13.8	119
NSW	8.154	74	83.6	9.6	13.0	120
VIC	6.614	56.0	63.85	7.85	14.0	120
QLD	5.322	43.9	51.1	7.2	16.4	135
SA	1.821	19	21.3	2.3	12.1	130
WA	2.785	20.8	23.2	2.4	11.5	86
TAS	0.572	6.2	7.0	0.8	12.9	140
NT	0.251	1.57	1.67	0.1	6.4	40
ACT	0.457	3.22	3.74	0.52	16.1	110

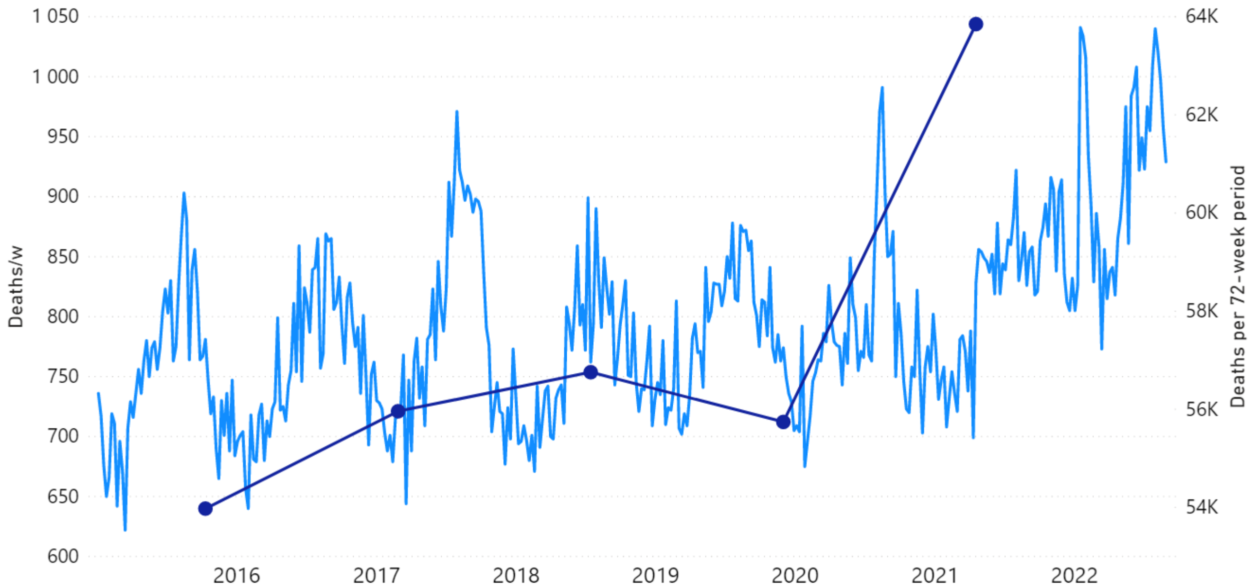
* The baseline-period 72-week-integrated mortality was estimated from an inspection of the values on the graphs (Figure A1-F1) for periods prior to the vaccination period, in such a way as to be representative of the value that would be predicted in the absence of the vaccination campaign and its effects.

Figure A1-F1 (containing 9 panels) follows.

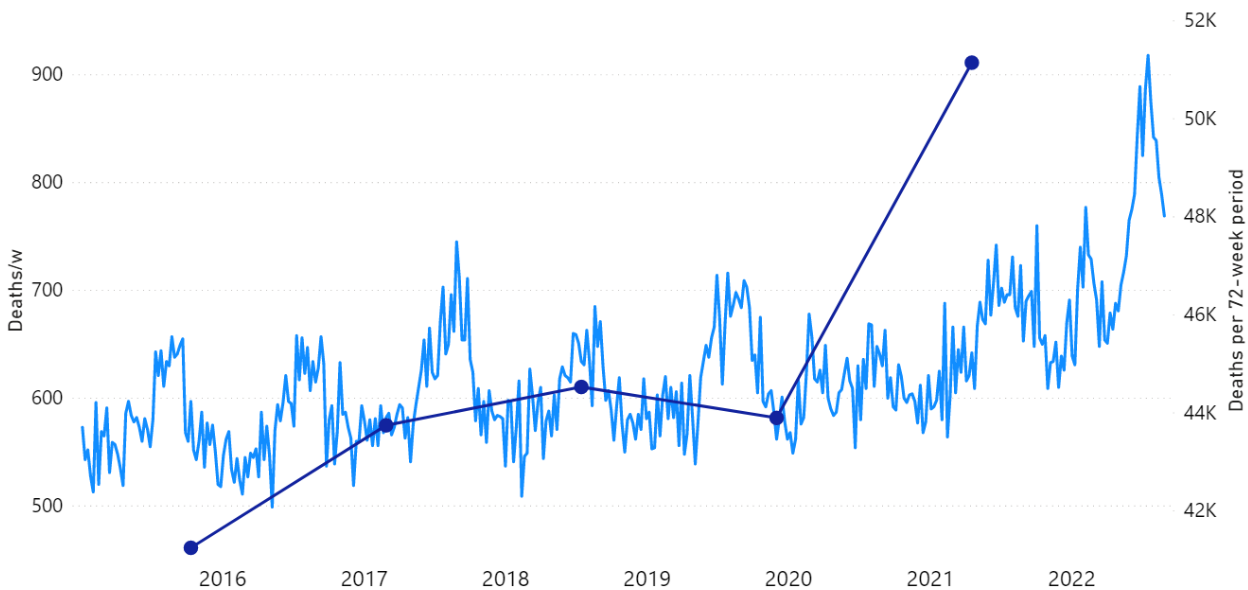


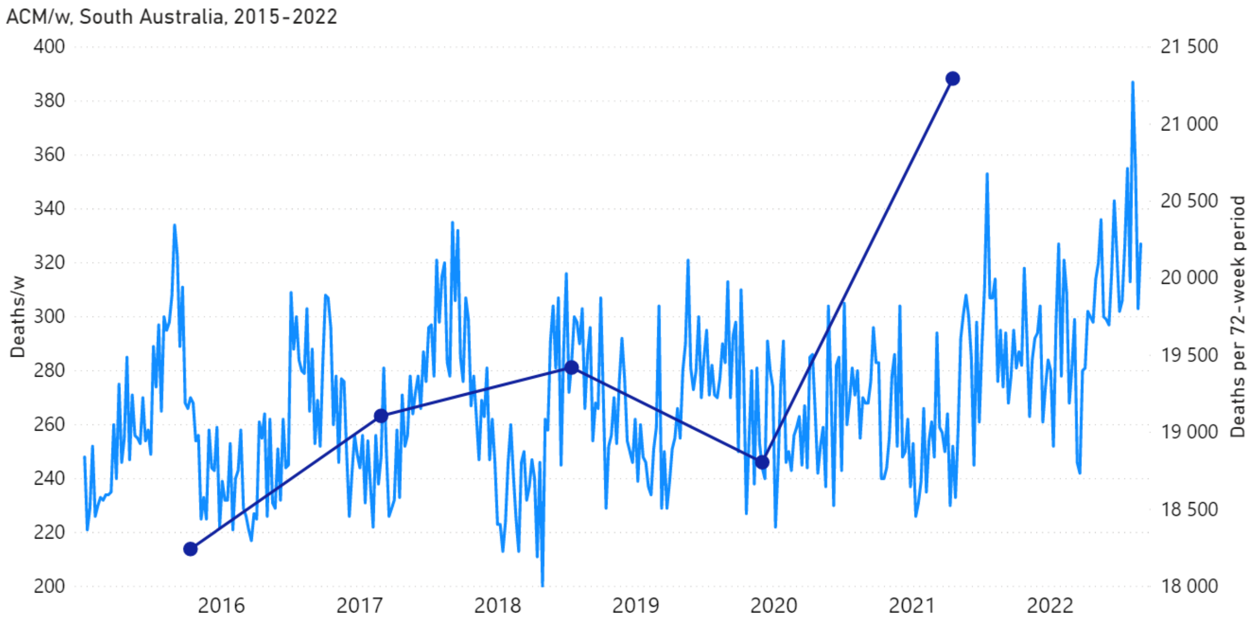


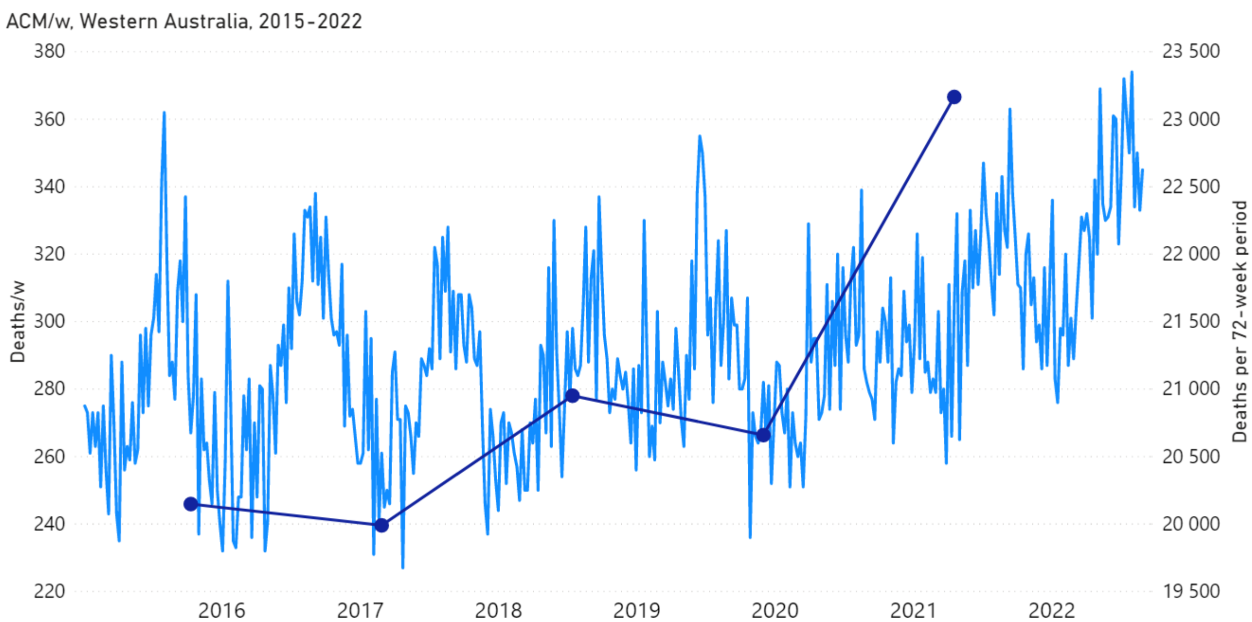
ACM/w, Victoria, 2015-2022

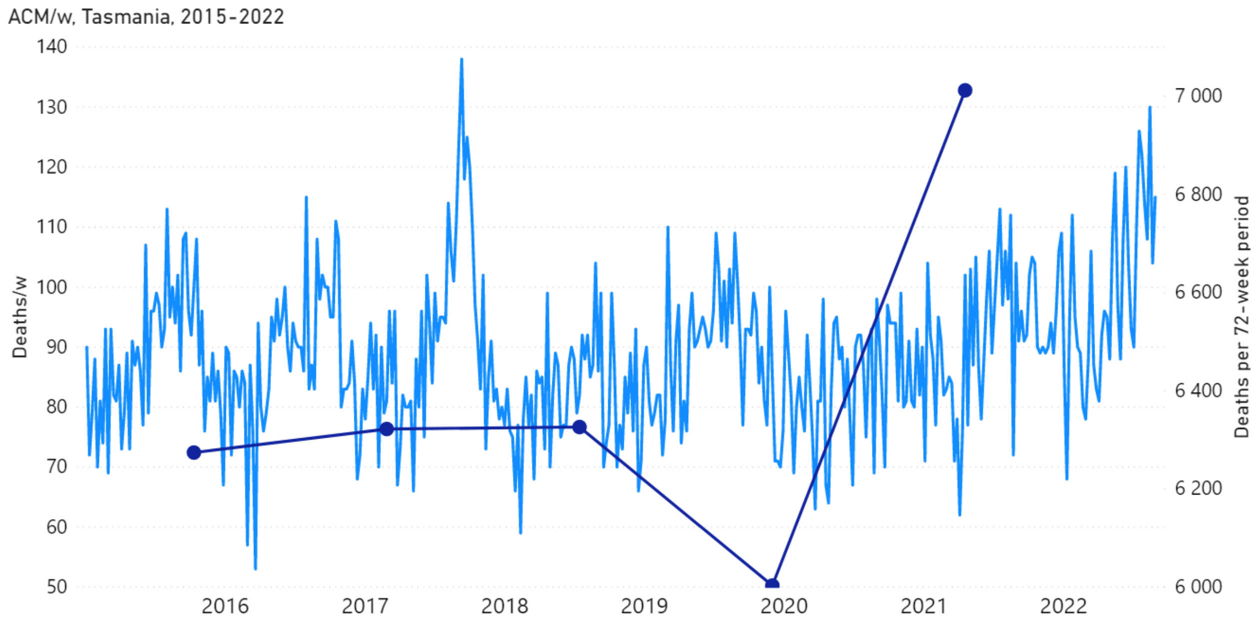


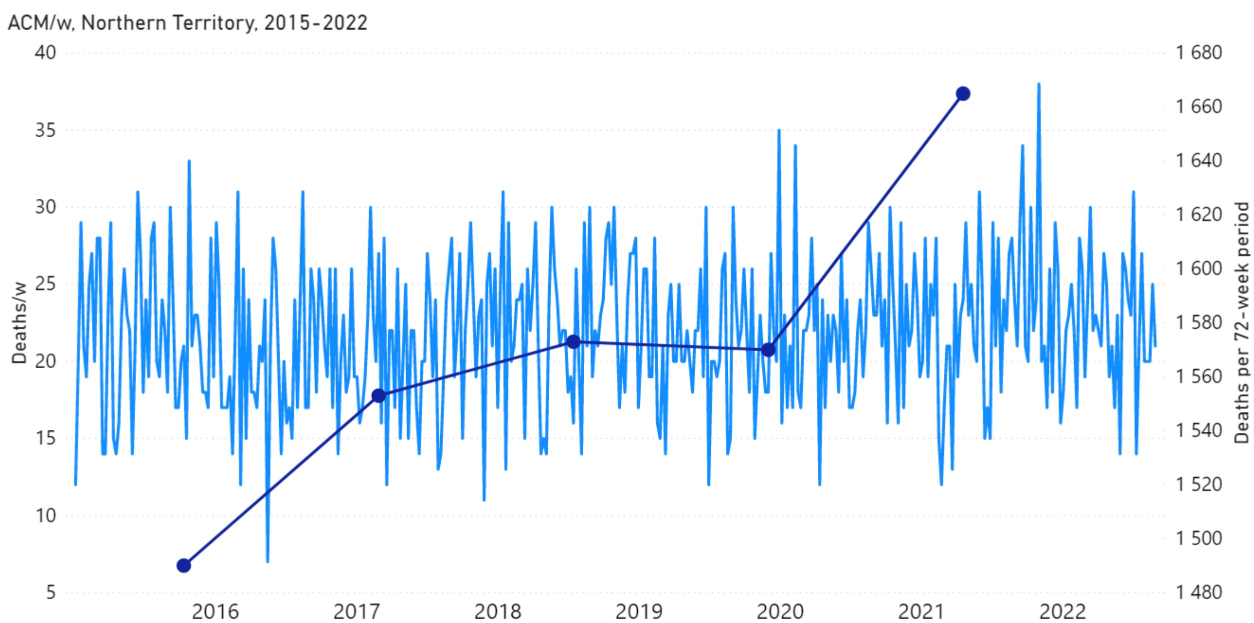
ACM/w, Queensland, 2015-2022

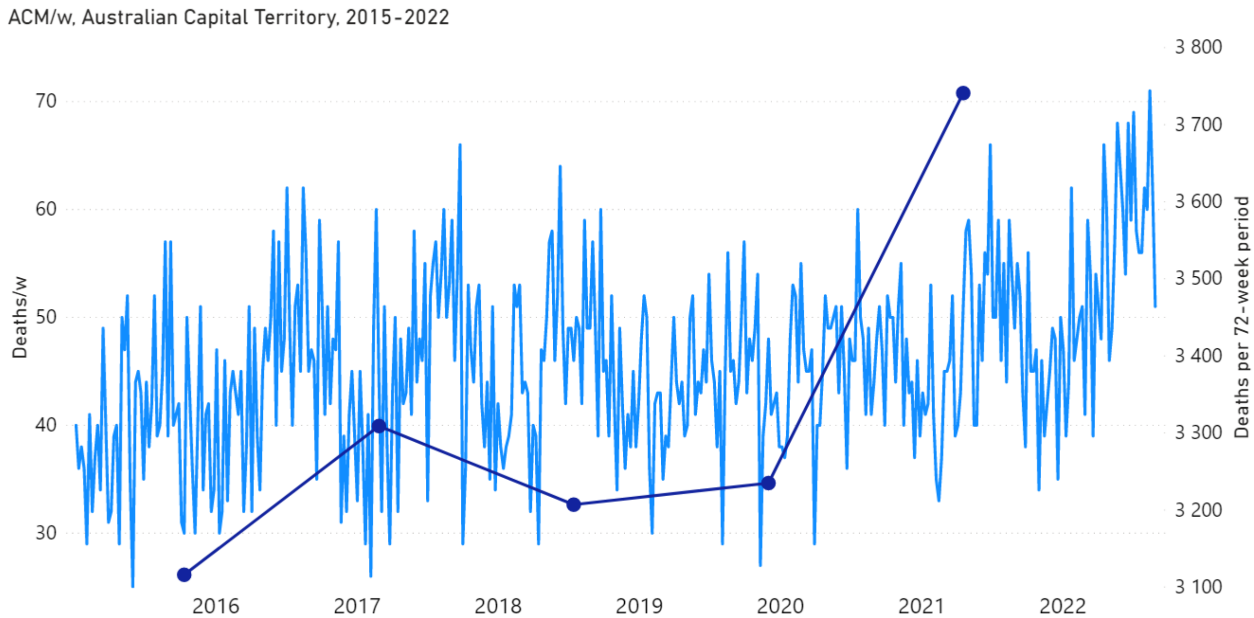



ACM/w, South Australia, 2015-2022
Deaths/w
Deaths per 72-week period
400
380
360
340
320
300
280
260
240
220
200
21 500
21 000
20 500
20 000
19 500
19 000
18 500
18 000
2016
2017
2018
2019
2020
2021
2022


ACM/w, Western Australia, 2015-2022
Deaths/w
Deaths per 72-week period
380
360
340
320
300
280
260
240
220
23 500
23 000
22 500
22 000
21 500
21 000
20 500
20 000
19 500
2016
2017
2018
2019
2020
2021
2022


ACM/w, Tasmania, 2015-2022
Deaths/w
Deaths per 72-week period
140
130
120
110
100
90
80
70
60
50
7 000
6 800
6 600
6 400
6 200
6 000
2016
2017
2018
2019
2020
2021
2022


ACM/w, Northern Territory, 2015-2022
Deaths/w
Deaths per 72-week period
40
35
30
25
20
15
10
5
1 680
1 660
1 640
1 620
1 600
1 580
1 560
1 540
1 520
1 500
1 480
2016
2017
2018
2019
2020
2021
2022


ACM/w, Australian Capital Territory, 2015-2022
Deaths/w
70
60
50
40
30
3 800
3 700
3 600
3 500
3 400
3 300
3 200
3 100
Deaths per 72-week period
2016
2017
2018
2019
2020
2021
2022

APPENDIX 2:
Mid-January to mid-February 2022 mortality peak not caused by a heatwave

This appendix is concerned with the question of whether the mid-January to mid-February 2022 prominent peak in all-cause mortality in Australia (occurring in NSW, VIC and QLD; see Appendix 1) can be due to a climatic heatwave.

It is important to address this question because sharp all-cause mortality peaks are often associated with exceptional summer heatwaves in mid-latitude countries (e.g. Rancourt et al., 2022, cited in the present article).

The most important heatwave to affect Eastern Australia over more than the last three decades was in 2009. The government report [Australian Government - Bureau of Meteorology, Special Climate Statement 17: The exceptional January-February 2009 heatwave in south-eastern Australia (issued 4 February 2009, updated 12 February 2009), http://www.bom.gov.au/climate/current/statements/scs17d.pdf , accessed 18 December 2022] describes it this way:

> "An exceptional heatwave affected south-eastern Australia during late January and early February 2009. The most extreme conditions occurred in northern and eastern Tasmania, most of Victoria and adjacent border areas of New South Wales, and southern South Australia, with many records set both for high day and night time temperatures as well as for the duration of extreme heat.
>
> There were two major episodes of exceptional high temperatures, from 28-31 January and 6-8 February, with slightly lower but still very high temperatures persisting in many inland areas through the period in between."

This exceptional 2009 heatwave did not cause any significant peak in all-cause mortality, as shown in Figure A2-F1, below. In fact, heatwaves essentially do not cause peaks in all-cause mortality in Australia, presumably because it's always hot in the

summers. Figure A2-F1 does not show any peaks, 1980-2022, which could be interpreted as summer heatwave peaks.

Also, there are no Australian Government, Bureau of Meteorology, Special Climate Statements (SCSs) 2006-2022, which can be interpreted to be associated with or similarly associated to the mid-January to mid-February 2022 prominent peak in all-cause mortality occurring in Eastern Australia (NSW, VIC, QLD) (see Appendix 1). See the list of SCSs here: http://www.bom.gov.au/climate/current/statements/ . Archived on 18 December 2022 here: https://archive.vn/WDlPA

And the Australian Government, Bureau of Meteorology, “Monthly Weather Review, Australia, January 2022” report [Product code IDCKGC1AR1. Prepared on 27 April 2022. http://www.bom.gov.au/climate/mwr/aus/mwr-aus-202201.pdf] makes no mention of any climate or weather event that could be associated with the mid-January to mid-February 2022 prominent peak in all-cause mortality occurring in Eastern Australia (NSW, VIC, QLD).

That the 2022 all-cause mortality peak of concern is not due to a heatwave is again corroborated by the fourteen maximum daily temperature maps for Australia shown below, for the years and dates as indicated on the maps.
[Source: http://www.bom.gov.au/climate/ . Specifically: http://www.bom.gov.au/jsp/awap/temp/rmse_archive.jsp?map=maxave&period=daily&year=2022&month=1&day=12]

The mid-January to mid-February 2022 prominent peak in all-cause mortality occurring in Eastern Australia (NSW, VIC, QLD) (see Appendix 1) — seen in Figure A2-F1 and in Figures 1, 2, 4A and 6 of the present article — is not due to any climate, weather or temperature event or anomaly.

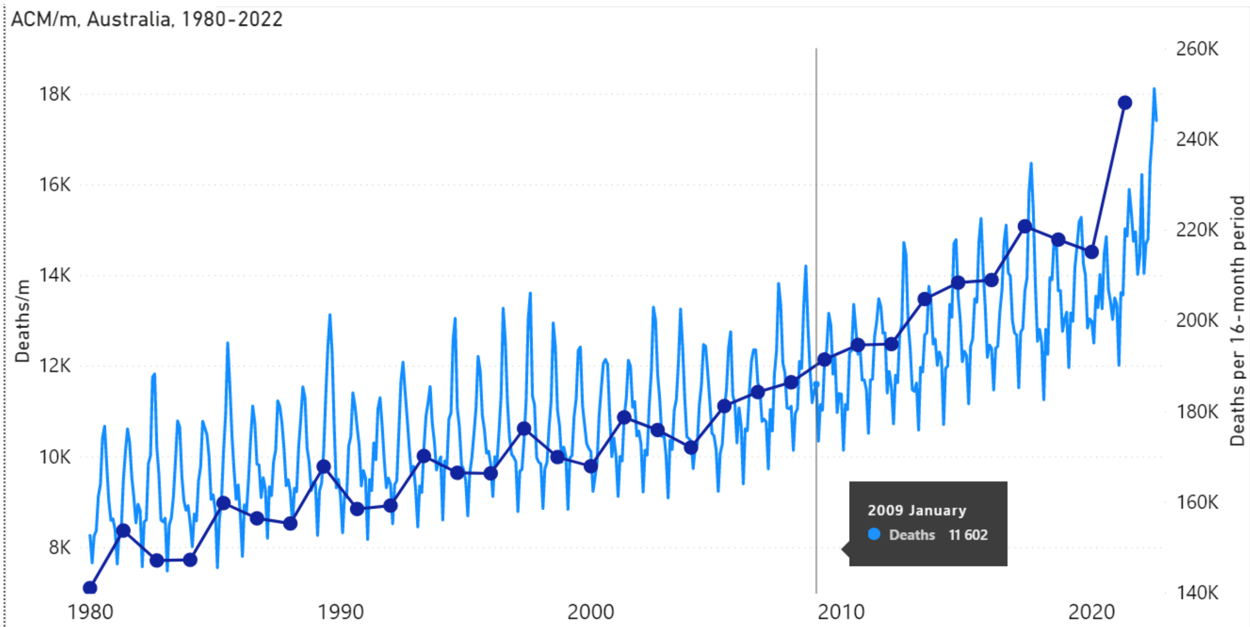
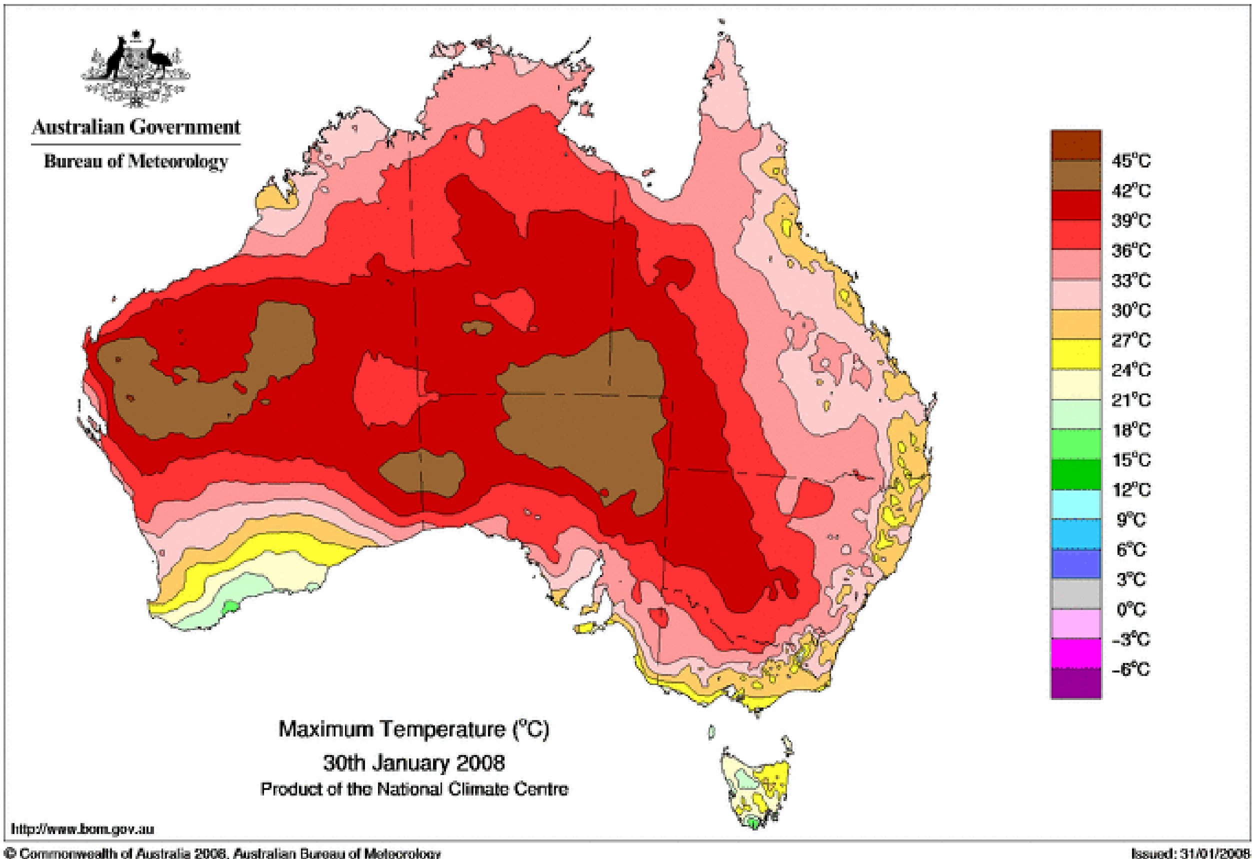
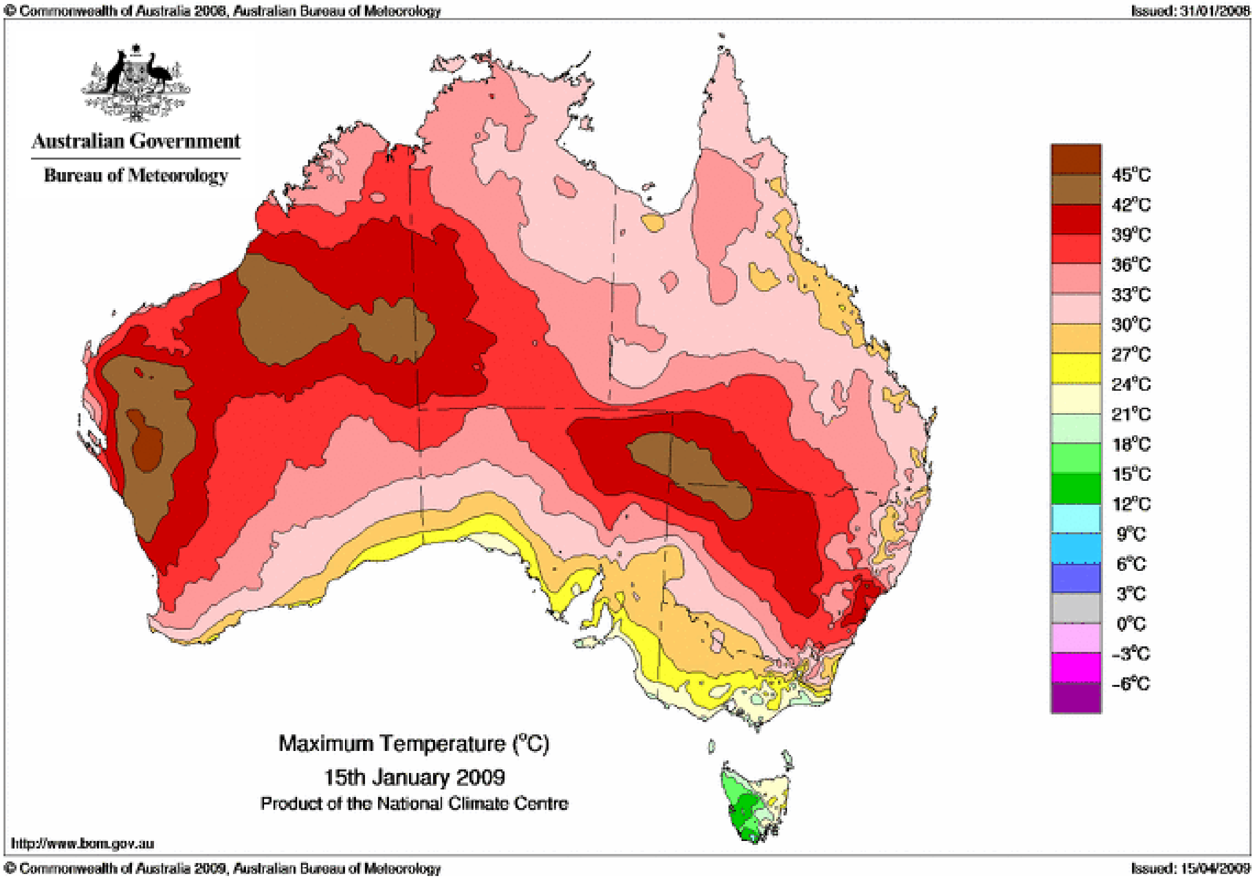
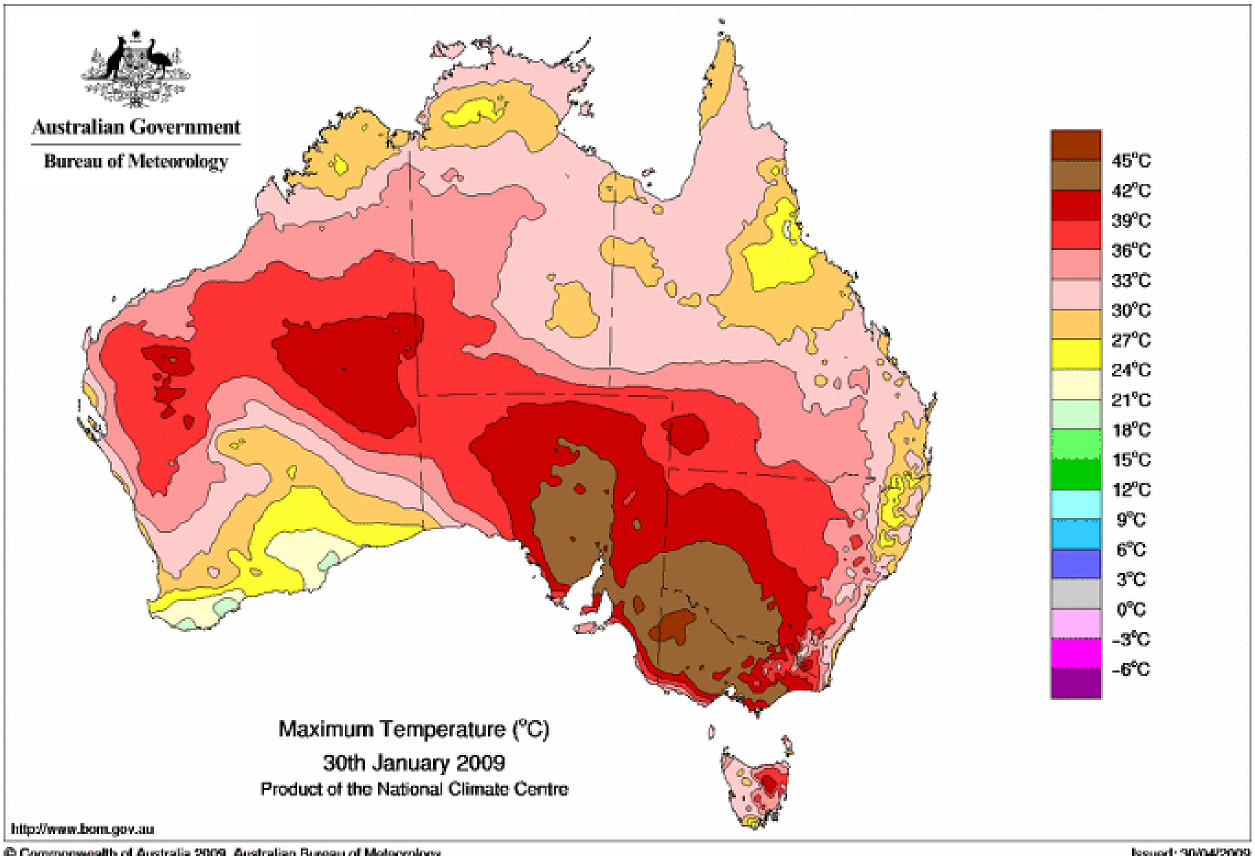
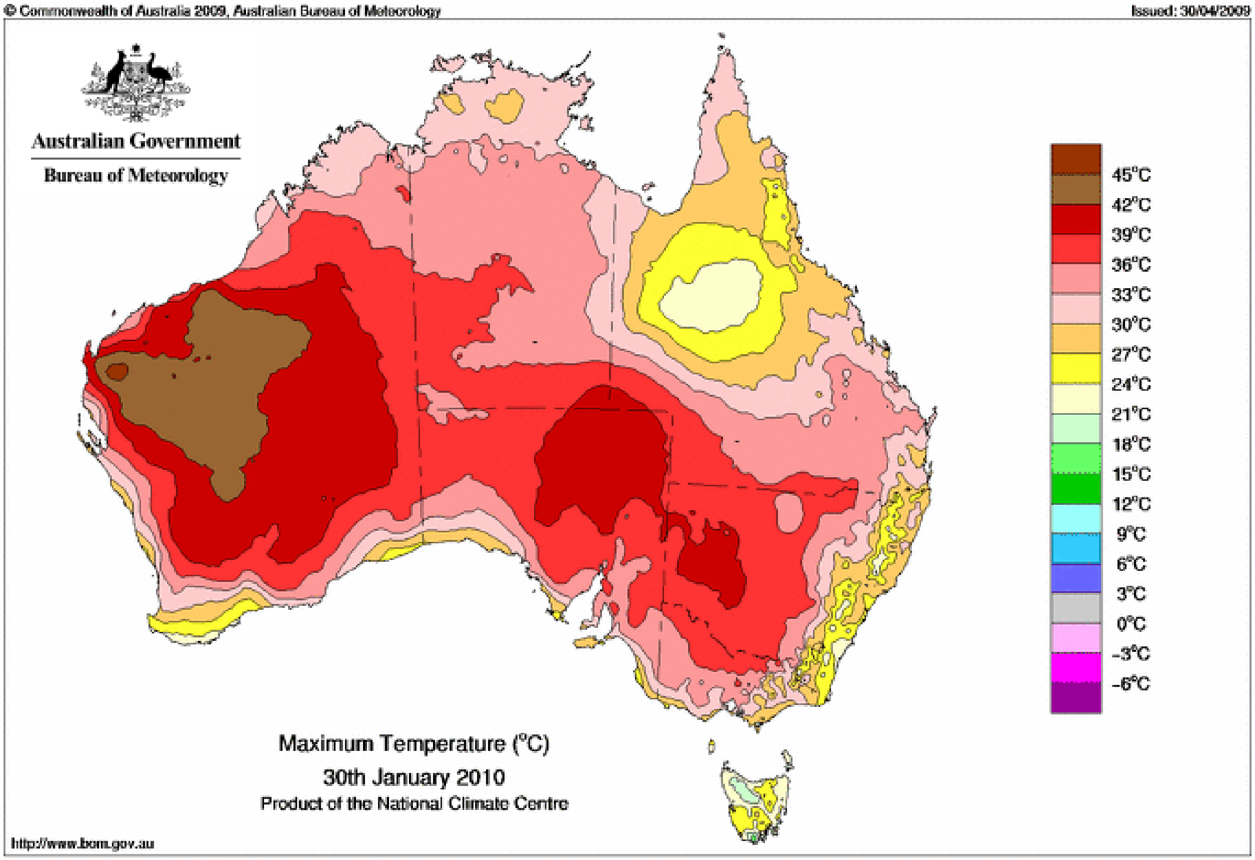


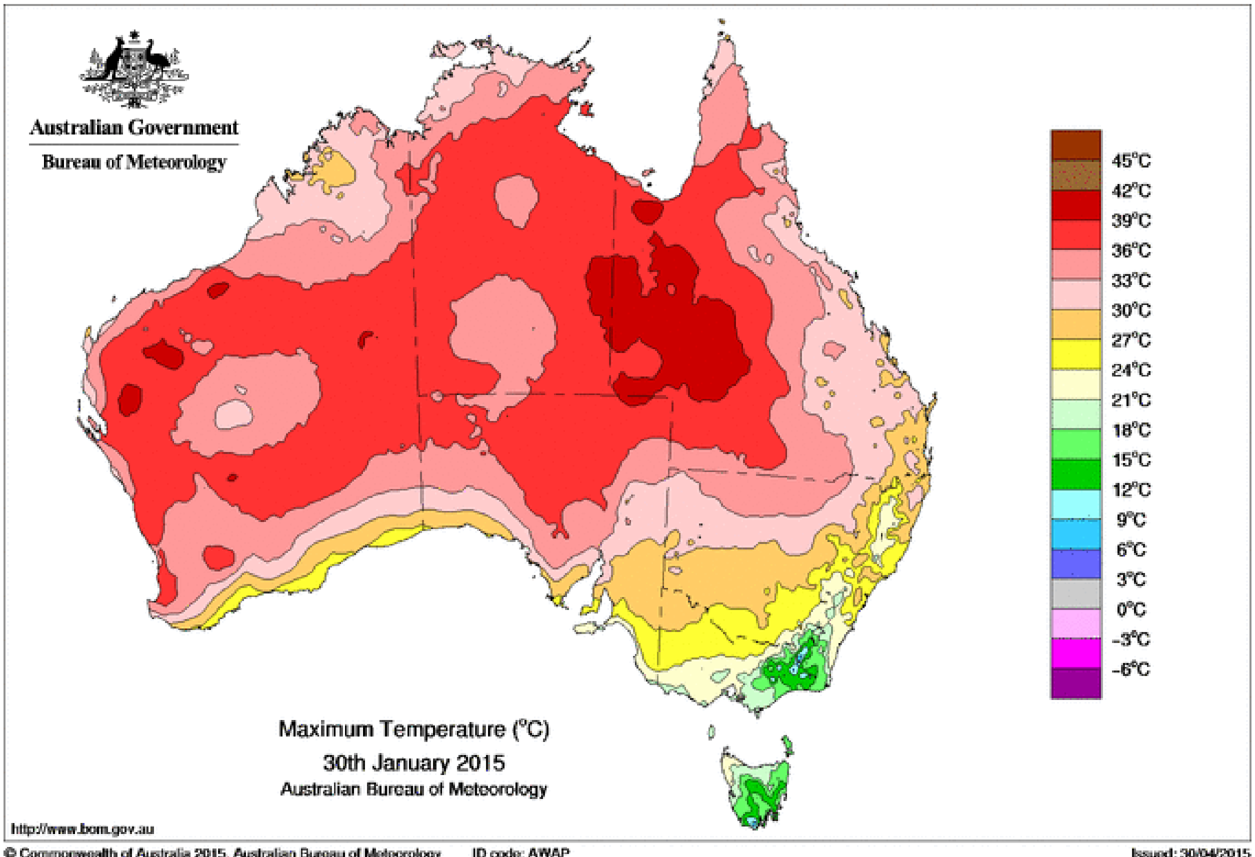
Figure A2-F1: All-cause mortality in Australia, all ages, from January 1980 through August 2022. Light-blue: All-cause mortality by month, left y-scale. Dark-blue: Integrated all-cause mortality over successive and non-overlapping 16-month periods (May 2021 through August 2022, for most recent period), right y-scale. Each point is positioned on the x-axis at the 1st month of its 16-month integration period. The labelled vertical line shows January 2009, which had a record-breaking heatwave and virtually no associated increase in mortality. February has lower mortality because it generally has only 28 days. (Data source: Australian Bureau of Statistics (2022a) for 2015-2022; United Nations (2022) for 1980-2014.)

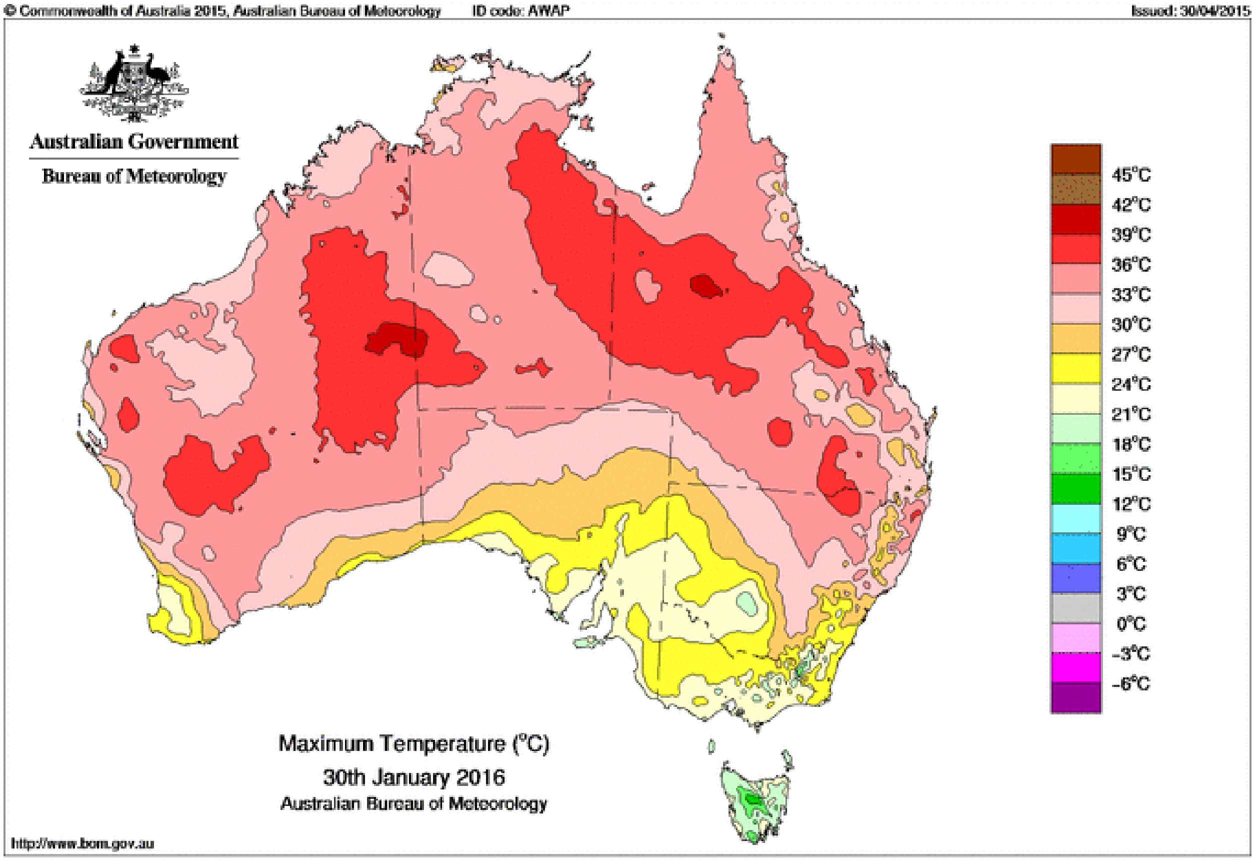

Australian Government
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-6°C
Maximum Temperature (°C)
30th January 2008
Product of the National Climate Centre
http://www.bom.gov.au
© Commonwealth of Australia 2008, Australian Bureau of Meteorology
Issued: 31/01/2008

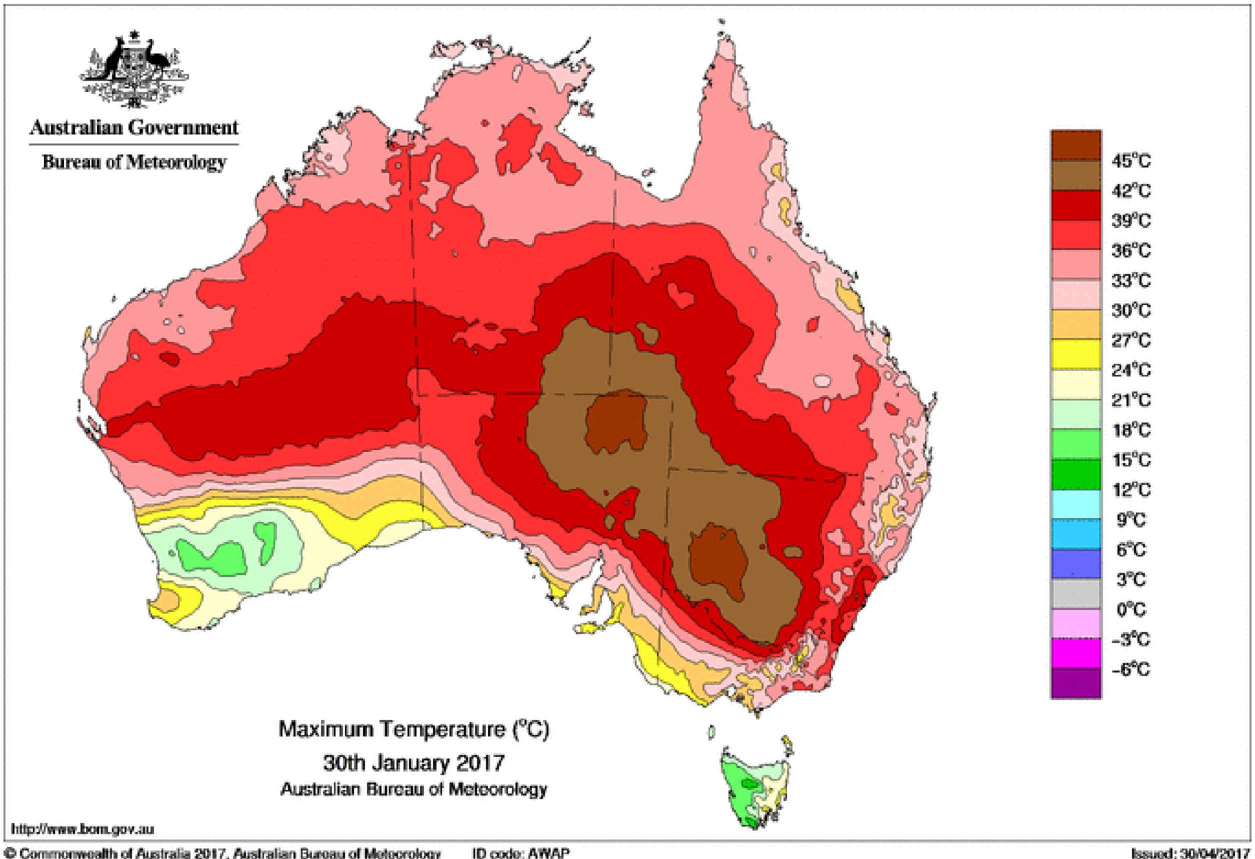

Australian Government
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Maximum Temperature (°C)
15th January 2009
Product of the National Climate Centre
http://www.bom.gov.au
© Commonwealth of Australia 2009, Australian Bureau of Meteorology
Issued: 15/04/2009

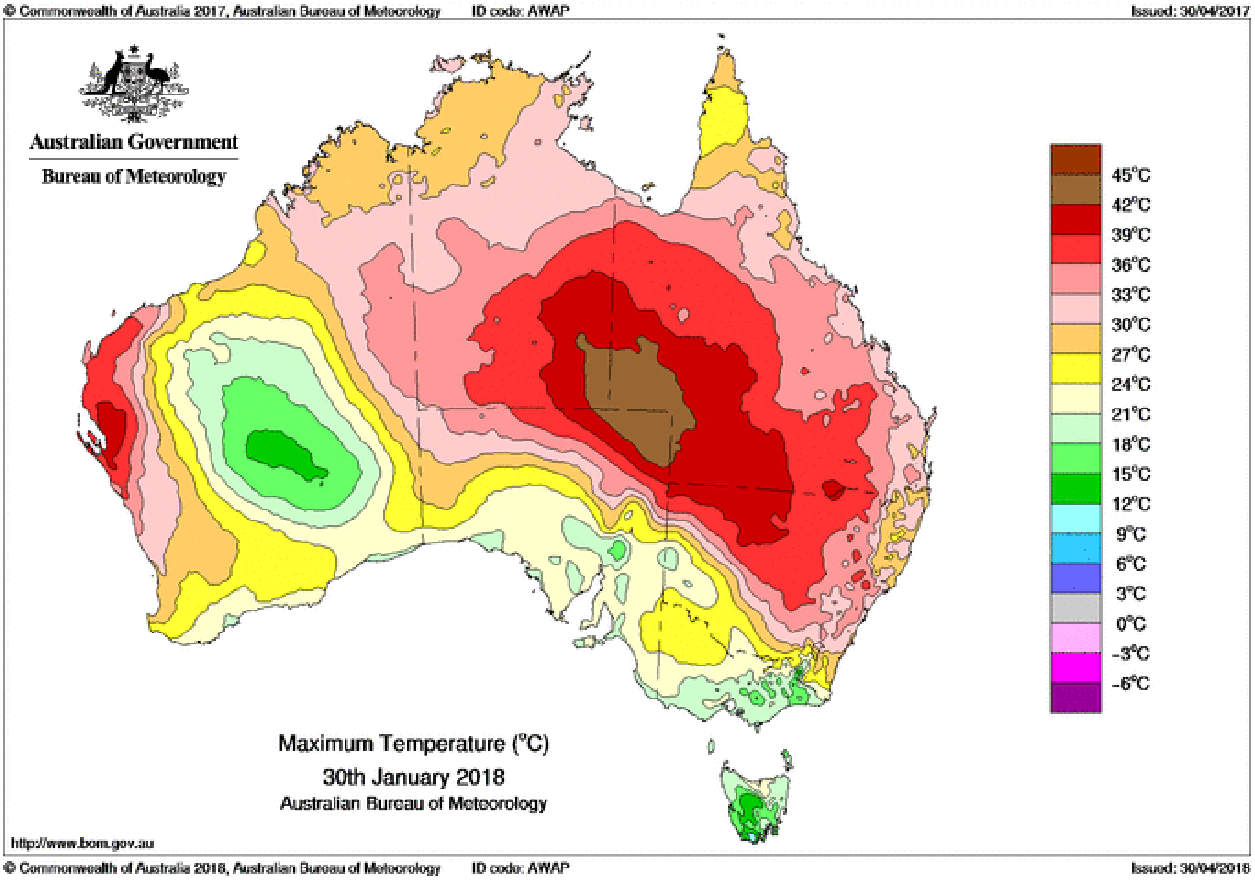

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Maximum Temperature (°C)
30th January 2009
Product of the National Climate Centre
http://www.bom.gov.au
© Commonwealth of Australia 2009, Australian Bureau of Meteorology
Issued: 30/04/2009

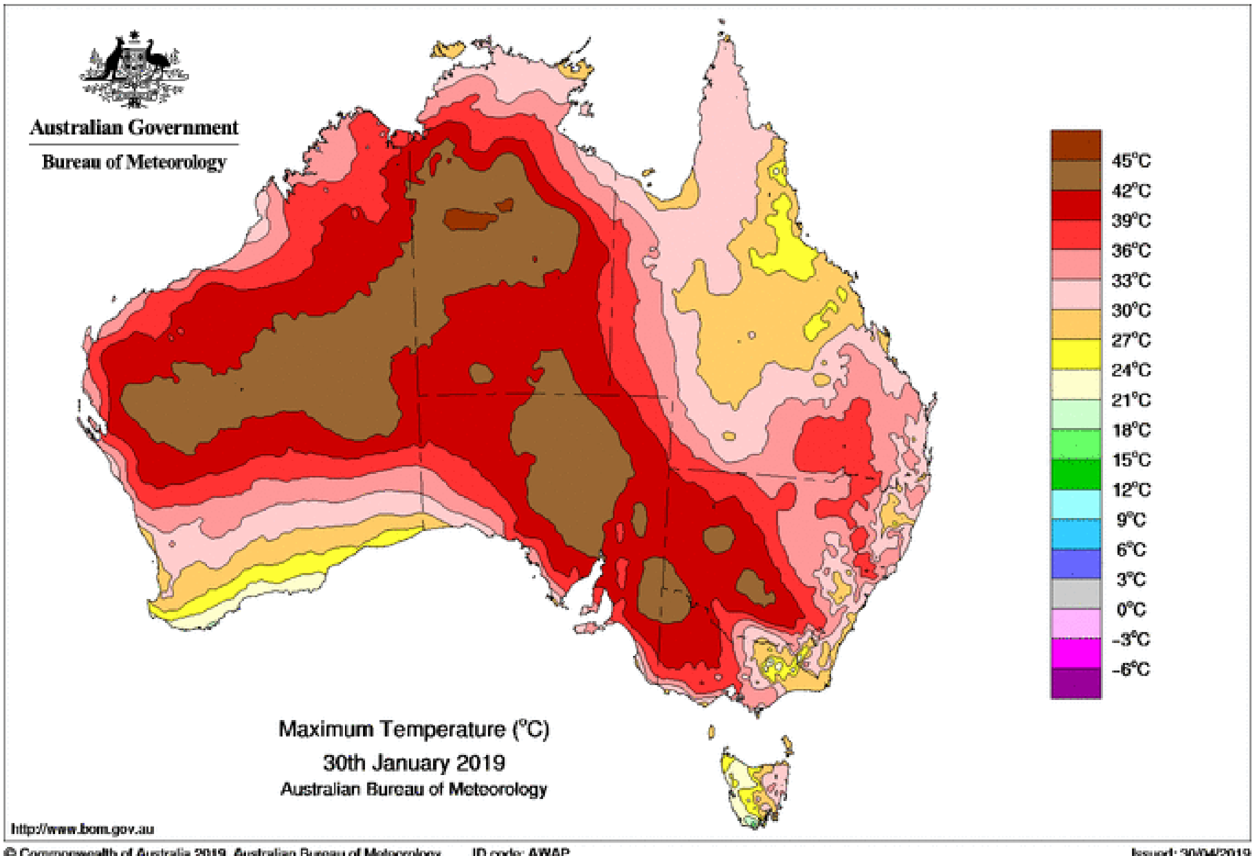

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Maximum Temperature (°C)
30th January 2010
Product of the National Climate Centre
http://www.bom.gov.au
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Issued: 30/04/2010


Australian Government
Bureau of Meteorology
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Maximum Temperature (°C)
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Australian Bureau of Meteorology
http://www.bom.gov.au
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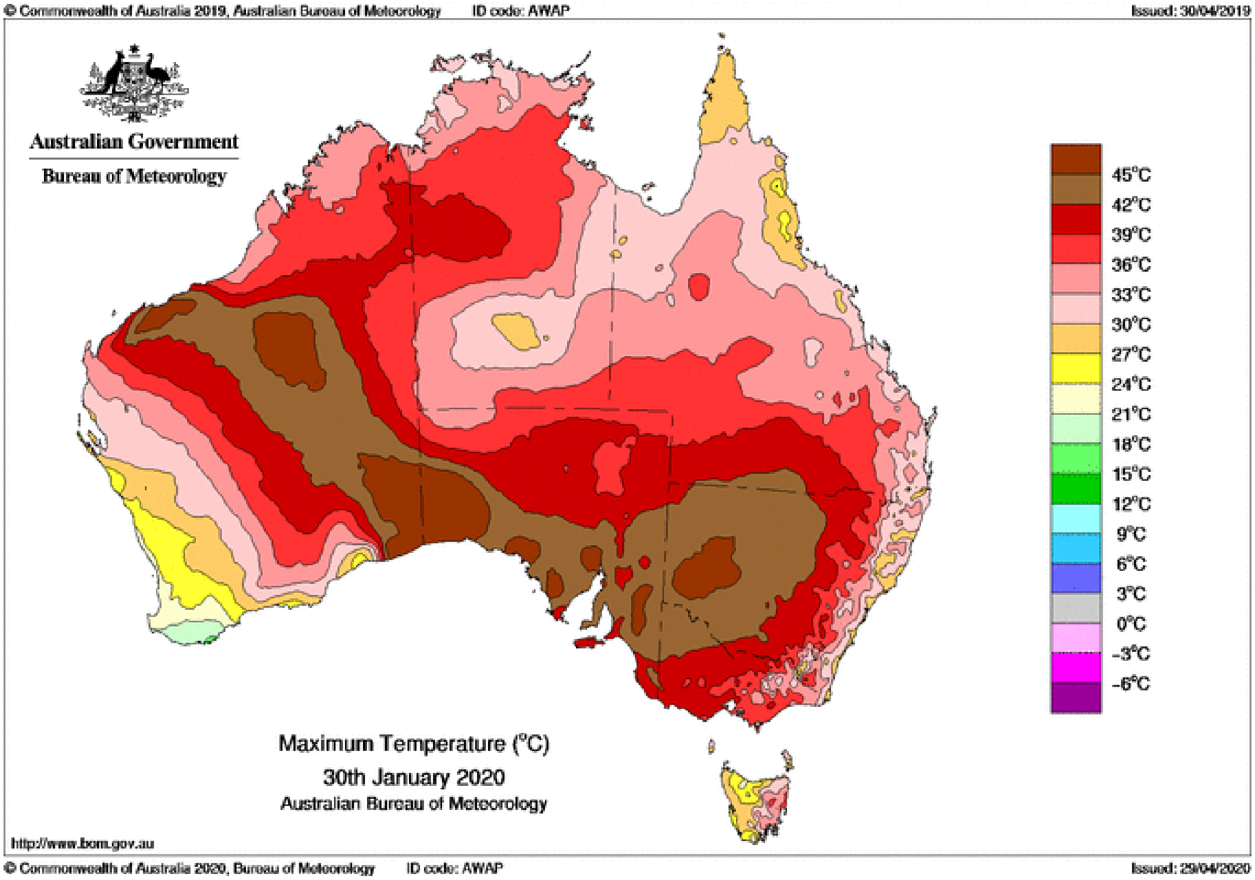

Australian Government
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Maximum Temperature (°C)
30th January 2016
Australian Bureau of Meteorology
http://www.bom.gov.au
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ID code: AWAP
Issued: 29/04/2016


Australian Government
Bureau of Meteorology
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Maximum Temperature (°C)
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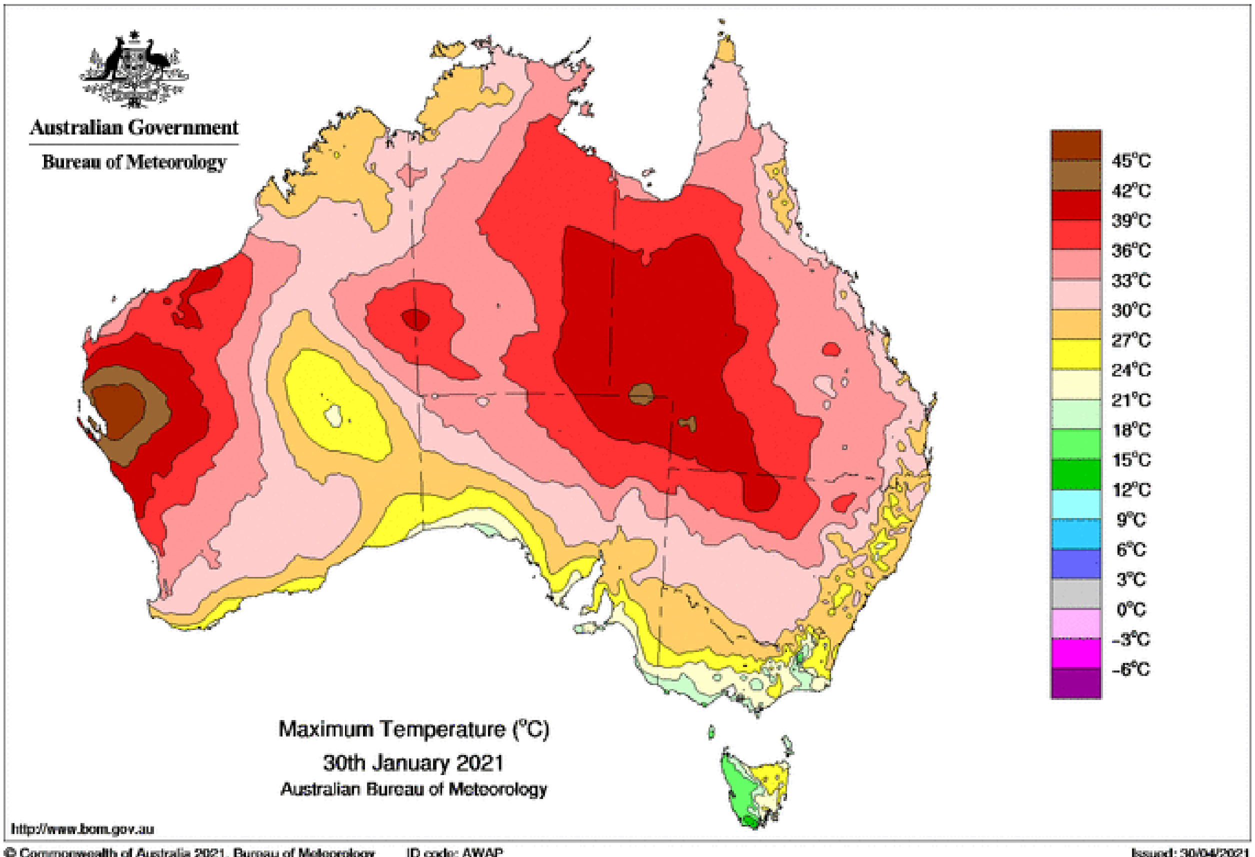

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Maximum Temperature (°C)
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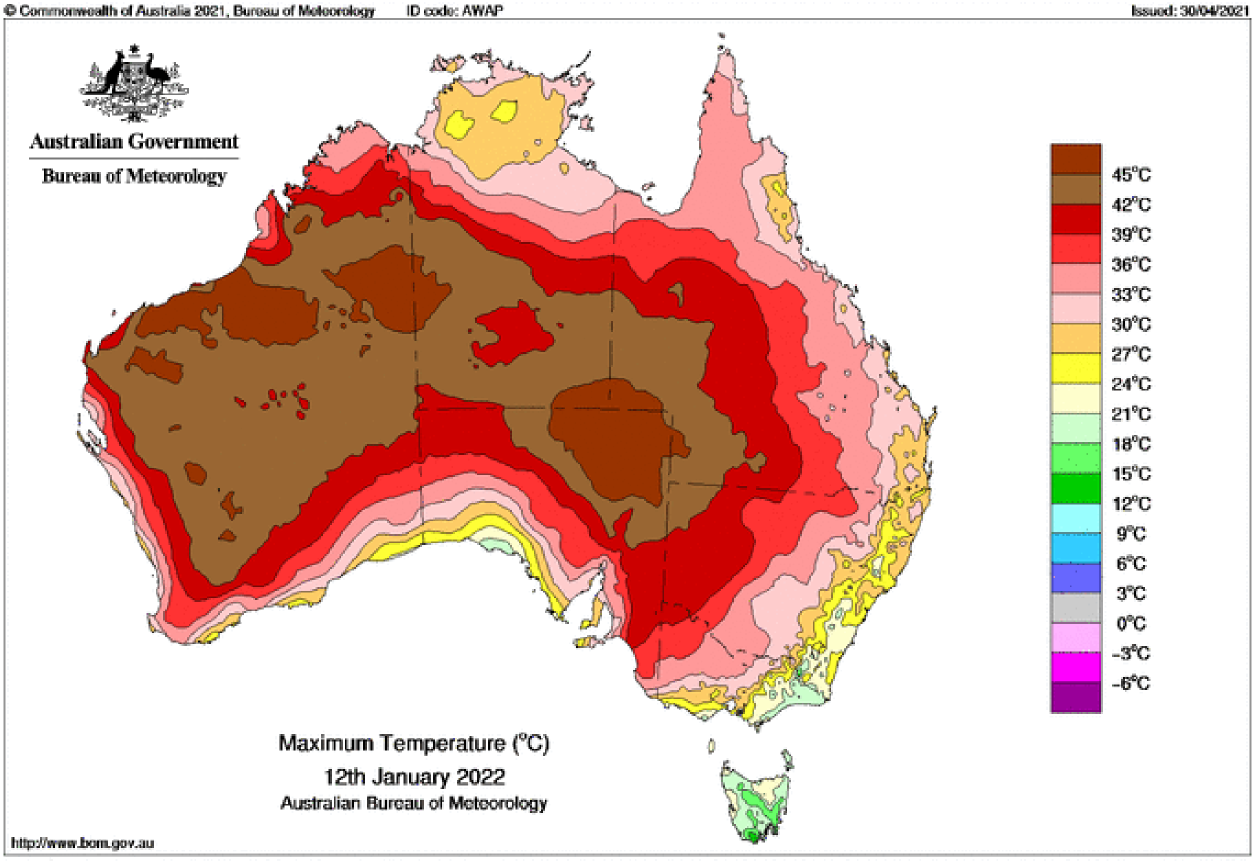


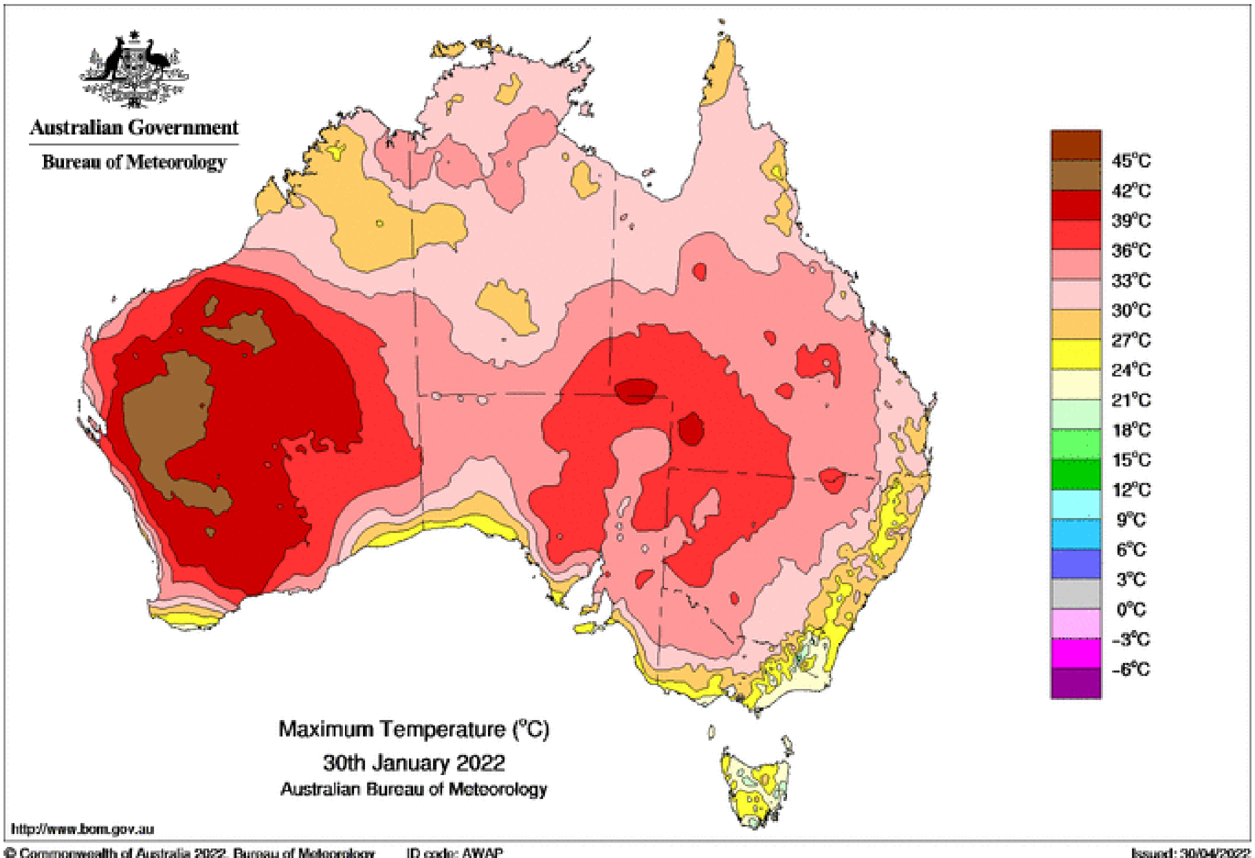
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Australian Bureau of Meteorology

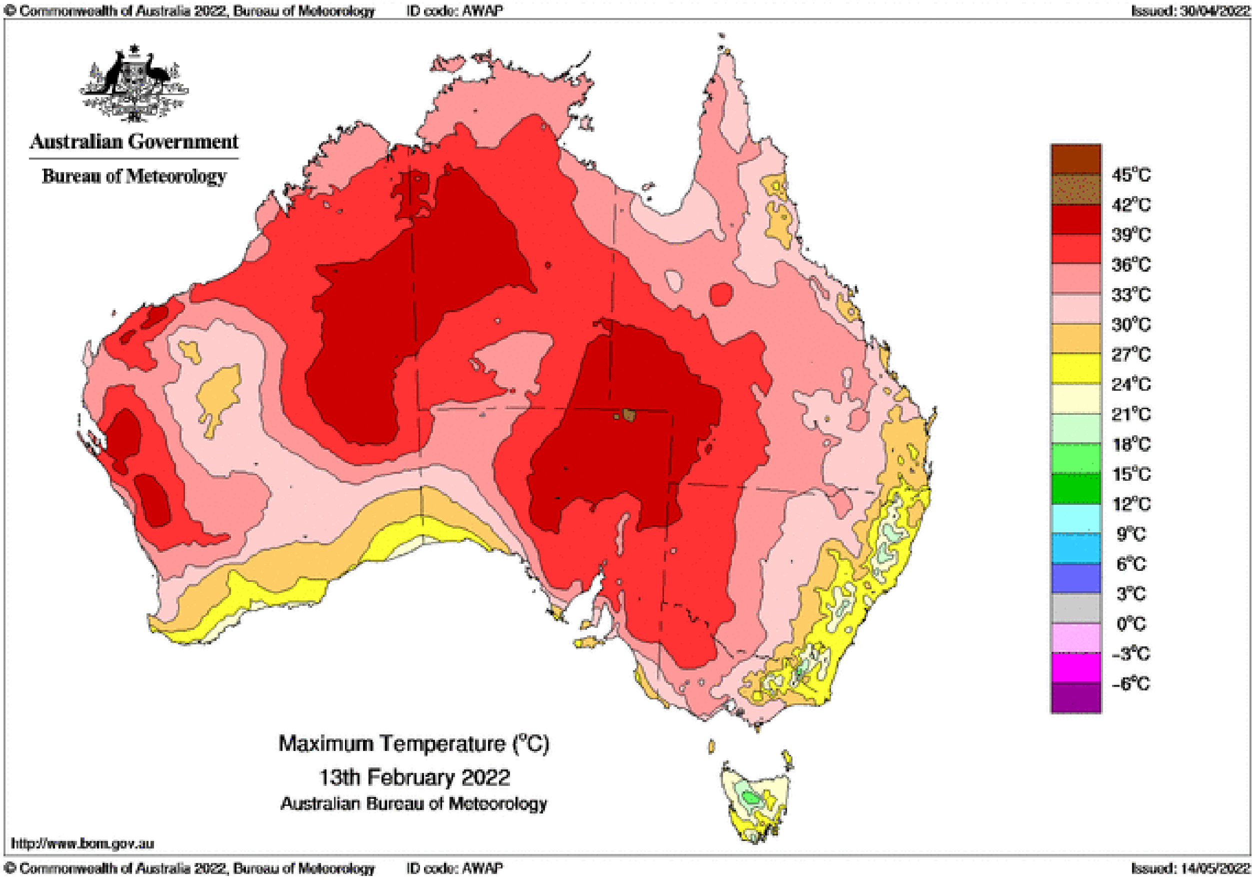


Maximum Temperature (°C)
30th January 2020
Australian Bureau of Meteorology


Australian Government
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Maximum Temperature (°C)
30th January 2021
Australian Bureau of Meteorology
http://www.bom.gov.au
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ID code: AWAP
Issued: 30/04/2021


Australian Government
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Maximum Temperature (°C)
12th January 2022
Australian Bureau of Meteorology
http://www.bom.gov.au
© Commonwealth of Australia 2022, Bureau of Meteorology
ID code: AWAP
Issued: 12/04/2022


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Maximum Temperature (°C)
30th January 2022
Australian Bureau of Meteorology
http://www.bom.gov.au
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ID code: AWAP
Issued: 30/04/2022


Australian Government
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Maximum Temperature (°C)
13th February 2022
Australian Bureau of Meteorology
http://www.bom.gov.au
© Commonwealth of Australia 2022, Bureau of Meteorology
ID code: AWAP
Issued: 14/05/2022

Brief Report |
6 December 2022

Probable causal association between India's extraordinary April-July 2021 excess-mortality event and the vaccine rollout

Denis G. Rancourt,* PhD

Correlation Research in the Public Interest
(correlation-canada.org)
* denis.rancourt@alumni.utoronto.ca

This report is simultaneously posted on several websites, including:
https://correlation-canada.org/
https://ocla.ca/
https://denisrancourt.ca/
https://archive.today/

ABSTRACT: India experienced a unique, sudden, unprecedented and extraordinarily large excess all-cause mortality event in April-July 2021, which is not adequately explained as a “second wave” or as being caused by a new variant of concern. After an overview of four recently published studies that have quantified the April-July 2021 excess all-cause mortality event, we give ten numbered arguments as to why we conclude that the extraordinary mortality event was caused by India’s vaccine rollout in its early stages. Therefore, it appears that the early rollout of the vaccine in India in April-July 2021 was devastating, causing the deaths of approximately 3.7 million residents, on administering approximately 350 million doses of the vaccine.

India experienced an extraordinary excess-mortality shock in April through July 2021, not seen in any other country in the world.

The mortality by week rose to almost 700% of its baseline value in April 2021, based on 90 municipalities in the state of Gujarat (Acosta et al. 2022; their Fig. 2), and the mortality by month rose to almost 400% of its baseline value in July 2021, based on 19 Indian states, 1.27 billion population (Leffler et al. 2022; their Fig. 1). To be clear, this represents all-cause mortalities that are 7-fold (by week) and 4-fold (by month) greater, respectively, than the pre-Covid (2019) all-cause mortalities in India.

This 4-month April-July 2021 excess mortality event in India is described in four independent studies published in leading medical journals (Acosta et al. 2022; Jha et al. 2022; Leffler et al. 2022; Lewnard et al. 2022); and it represents the great majority of excess all-cause deaths for the entire Covid period examined since a pandemic was declared by the World Health Organization on 11 March 2020.

Given the extraordinary characteristics of the 4-month April-July 2021 excess mortality event in India, it is useful to reproduce key figures from the said studies, in order to grasp its significance and nature, as follows.

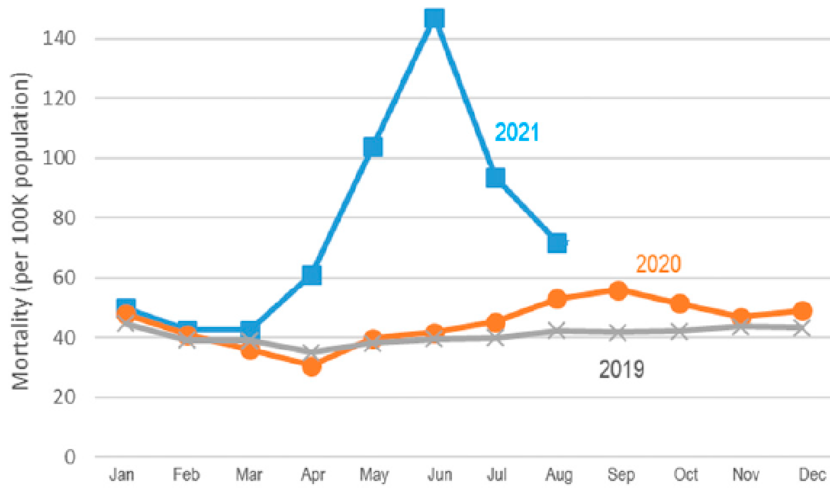


FIGURE 1. Per capita all-cause mortality in India by month, 2019 to 2021, based on 13 states and two union territories, as described in the Methods section. This figure appears in color at www.ajtmh.org.

Figure 1: Leffler et al. (2022), using 19 Indian states, 1.27 billion population, their Fig. 1.

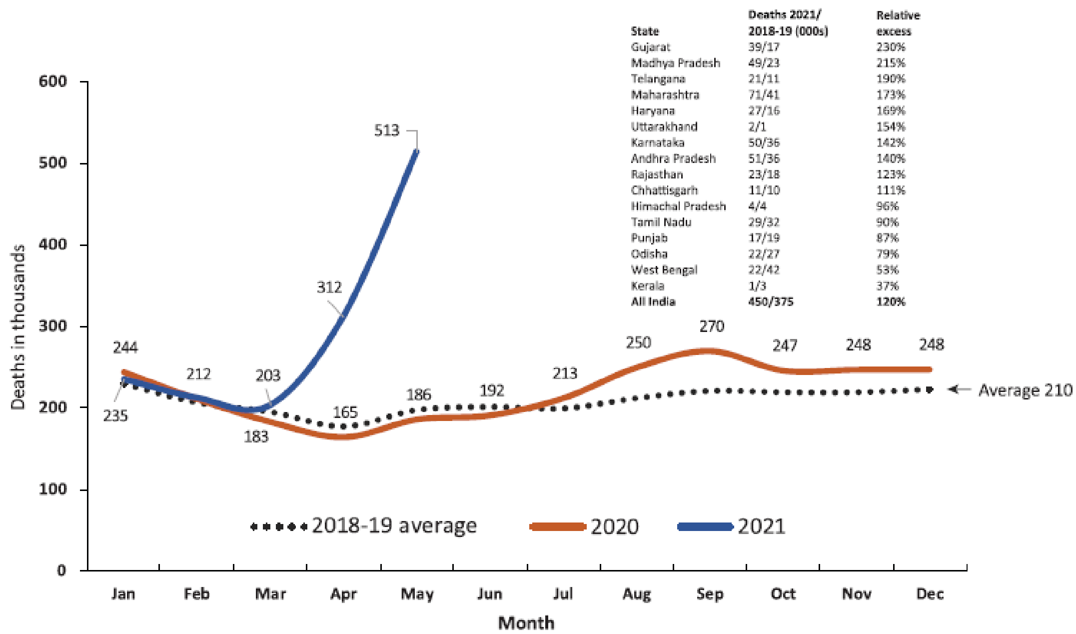


Fig. 3. Reported deaths from all causes in India's Ministry of Health and Family Welfare Management Information System covering 0.2 million health facilities nationally, 2020 and 2021, versus average of 2018–2019, by month. The inset shows the increases in selected states and nationally for the April–May 2021 relative to the 2018–2019 averages for the same months of comparison. Table S6 provides the input data.

Figure 2: Jha et al. (2022), using 0.2 million health facilities nationally, their Fig. 3. This is essentially the same figure as Fig. 1 in Deshmukh et al. (2021).

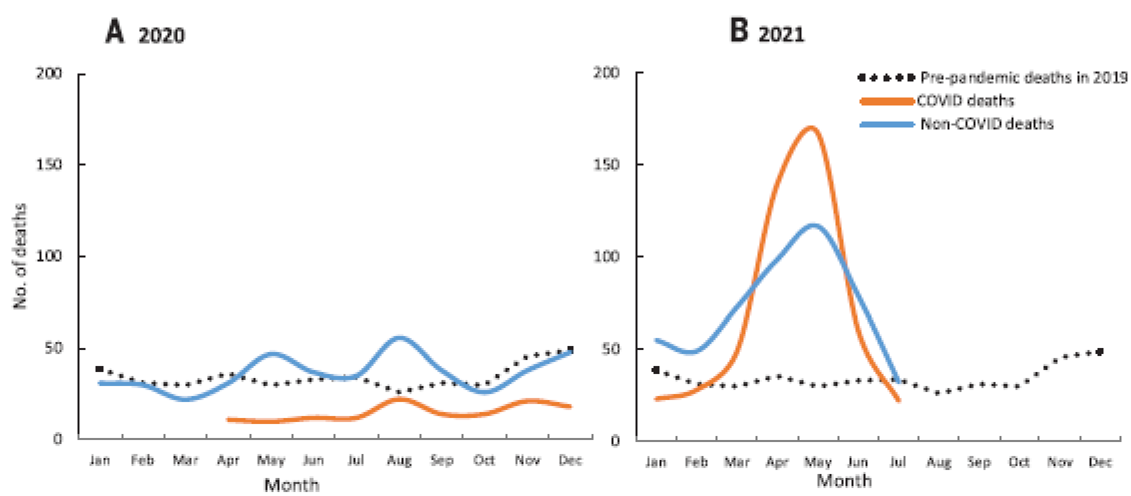


Fig. 2. Monthly reporting of deaths as COVID (including COVID-associated) and non-COVID by month for 2019 to 2021 in a substudy of 57,000 adults in 13,500 households within the COVID Tracker survey (*2*). Table S3 provides the input data. (**A**) 2020 deaths; (**B**) 2021 deaths.

Figure 3: Jha et al. (2022), using a survey study of 57 thousand adults, their Fig. 2.

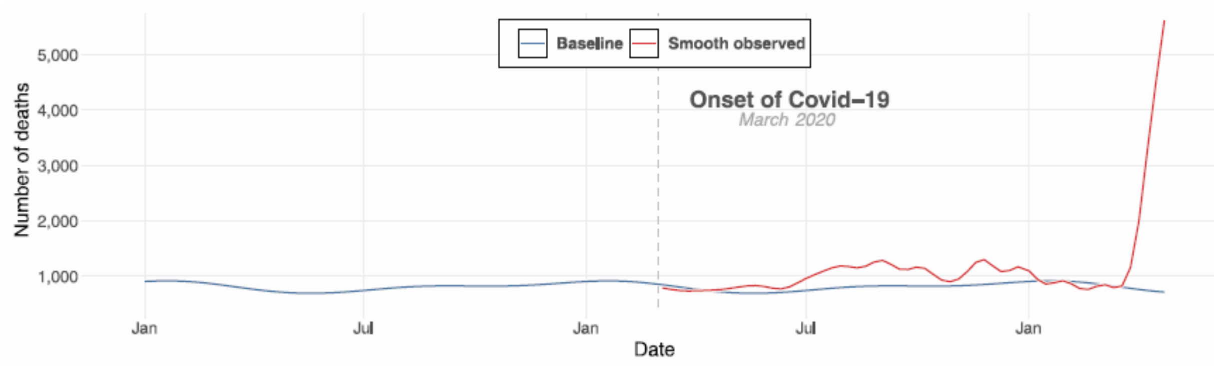


Fig 1. Model fit for weekly death counts. Model fit for weekly death counts amalgamated from multiple municipalities in Gujarat, India. The gray data points are weekly death counts, the dashed-vertical line represents the onset of Covid-19, the blue curve represents the expected weekly death counts based on historical data, and the red curve represents the smooth observed weekly death counts during Covid-19.

https://doi.org/10.1371/journal.pgph.0000824.g001

Figure 4: Acosta et al. (2022), using death certificates from 90 municipalities in the Indian state of Gujarat, their Fig. 1.

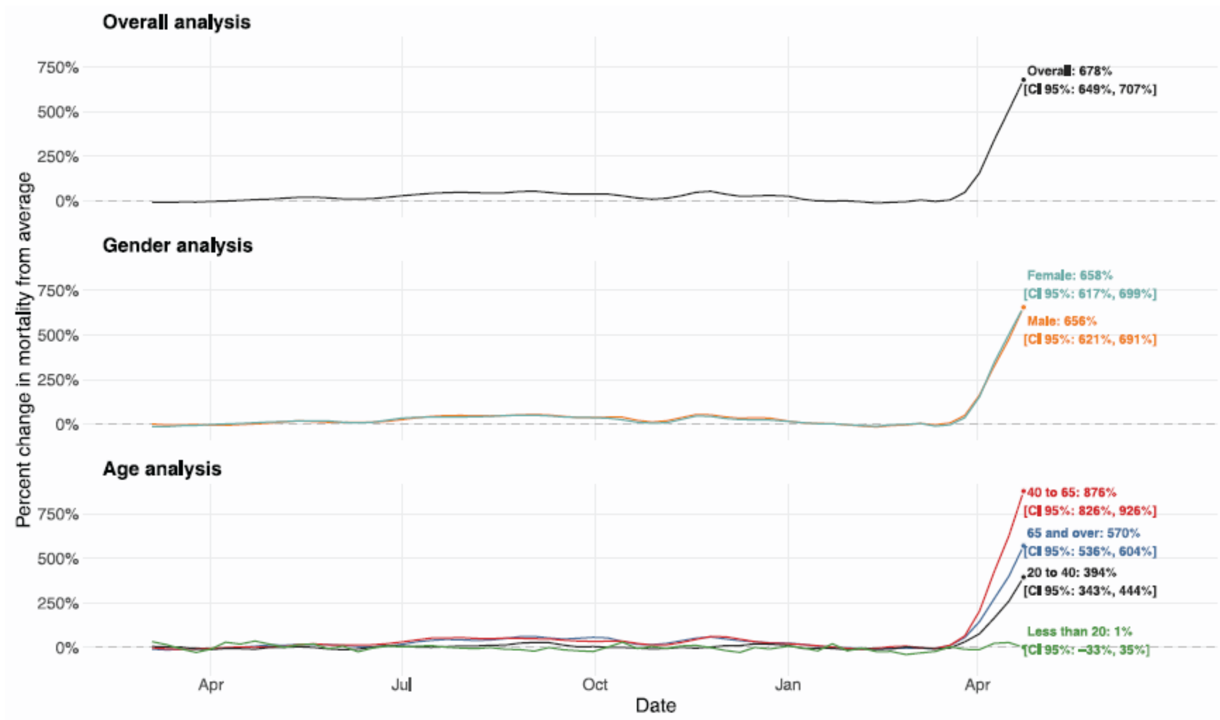


Fig 2. Estimated percent change in mortality from average in Gujarat, India, from March 2020 to April 2021. Estimated percent change in mortality from average in Gujarat, India, from March 2020 to April 2021. The solid-curves represent percent changes from average mortality for each group. 95% confidence intervals were omitted for better readability. The point estimate and corresponding 95% confidence intervals for April 16, 2021, the week of peak excess mortality, are displayed in text on the right and highlighted with a data point.

https://doi.org/10.1371/journal.pgph.0000824.g002

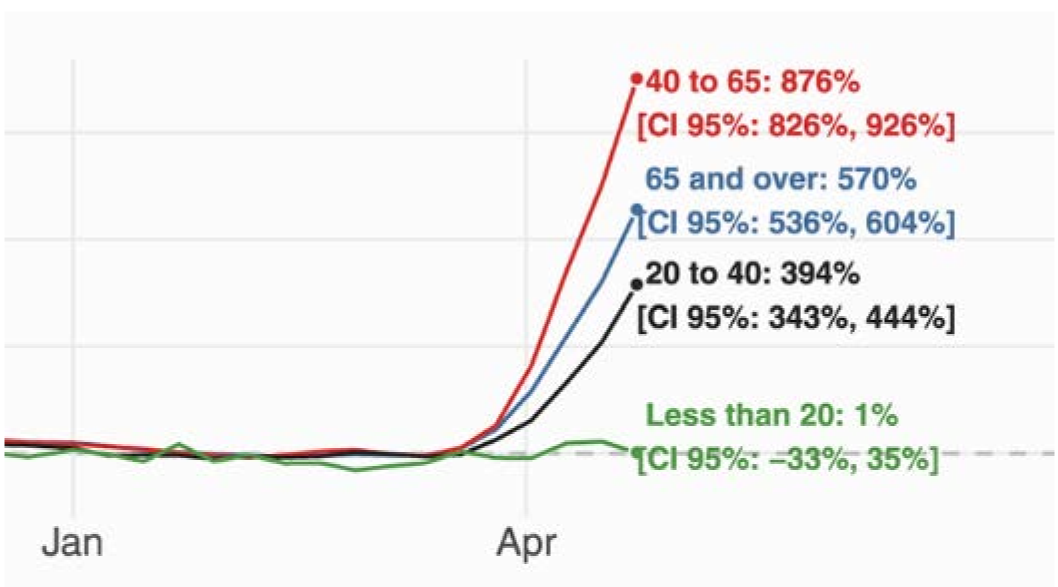


Figure 5: Acosta et al. (2022), using death certificates from 90 municipalities in the Indian state of Gujarat, their Fig. 2. Based on mortality by week. (Upper) Full figure. (Lower) Selected enlargement.

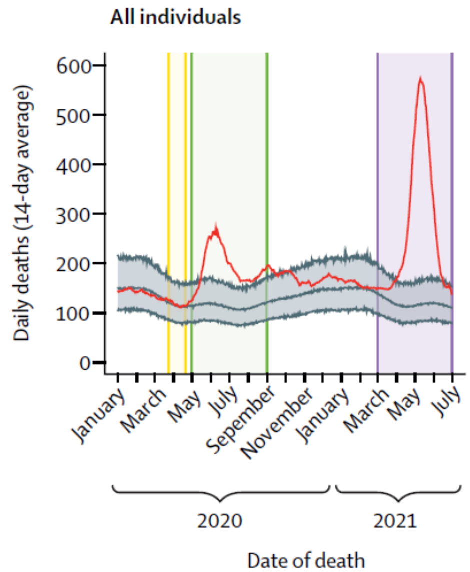


Figure 6: Lewnard et al. (2022), in the Chennai district, India, their Fig. 1. (Red line: 14-day moving average estimates of daily mortality in 2020 and 2021 (observed deaths), corrected for lagged reporting based on 2019 observations.)

When such a large, unique, and sudden feature in mortality of all causes occurs in any jurisdiction, it demands thorough investigation, if the cause is not empirically obvious, such as a massive earthquake or a genocidal military attack. This holds even during a declared pandemic, given the unique, sudden, unprecedented and large-magnitude nature of the event in India.

All of the above-cited authors who have reported on the 4-month April-July 2021 excess mortality event in India have referred to the event as being India's "second wave" and have used their all-cause mortality evaluations to infer that COVID-19 mortality is potentially largely underestimated by India's official Covid-death statistics.

In this author's opinion, if that was India's "second wave" then, by comparison, India virtually did not have a "first wave", and essentially did not have a death-causing pandemic prior to April 2021.

None of the above-cited authors who have reported on the 4-month April-July 2021 excess mortality event in India have mentioned the remarkable coincidence that the said excess mortality event coincides in time with India’s vaccine rollout, starting on 1 March 2021 with those 60 years and older and those over 45 years and having “comorbidities” (among 20 listed comorbidities) (*The Economic Times*, 24 February 2021; Ministry of Health and Family Welfare, Government of India, 2021), extended to all residents over 45 years on 1 April 2021; and coinciding in time with the government’s 4-day *Teeka Utsav* ("Vaccine Festival") from 11 to 14 April 2021, in which some 100 million vaccine doses were administered by its completion: “Elderly people or those who may not be much educated should be helped in getting the vaccine”, Prime Minister Modi said (*Mint*, 11 April 2021).

To appreciate India’s vaccine rollout, its official statistics are a reference, as follows.

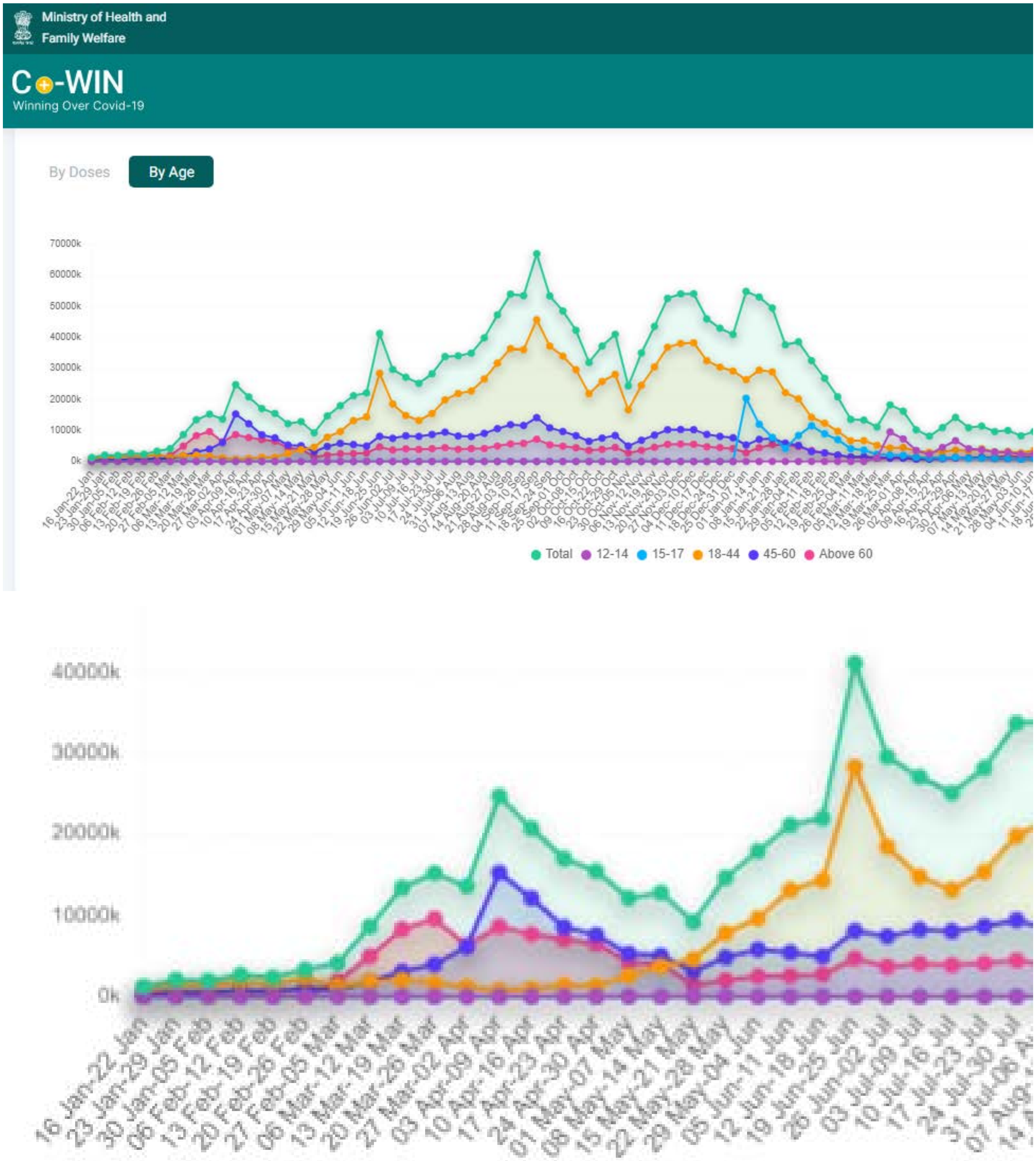


Figure 7: Ministry of Health and Family Welfare, Government of India (2022): C+ WIN dashboard, by age as indicated. (Upper) Broad view. (Lower) Selected enlargement.

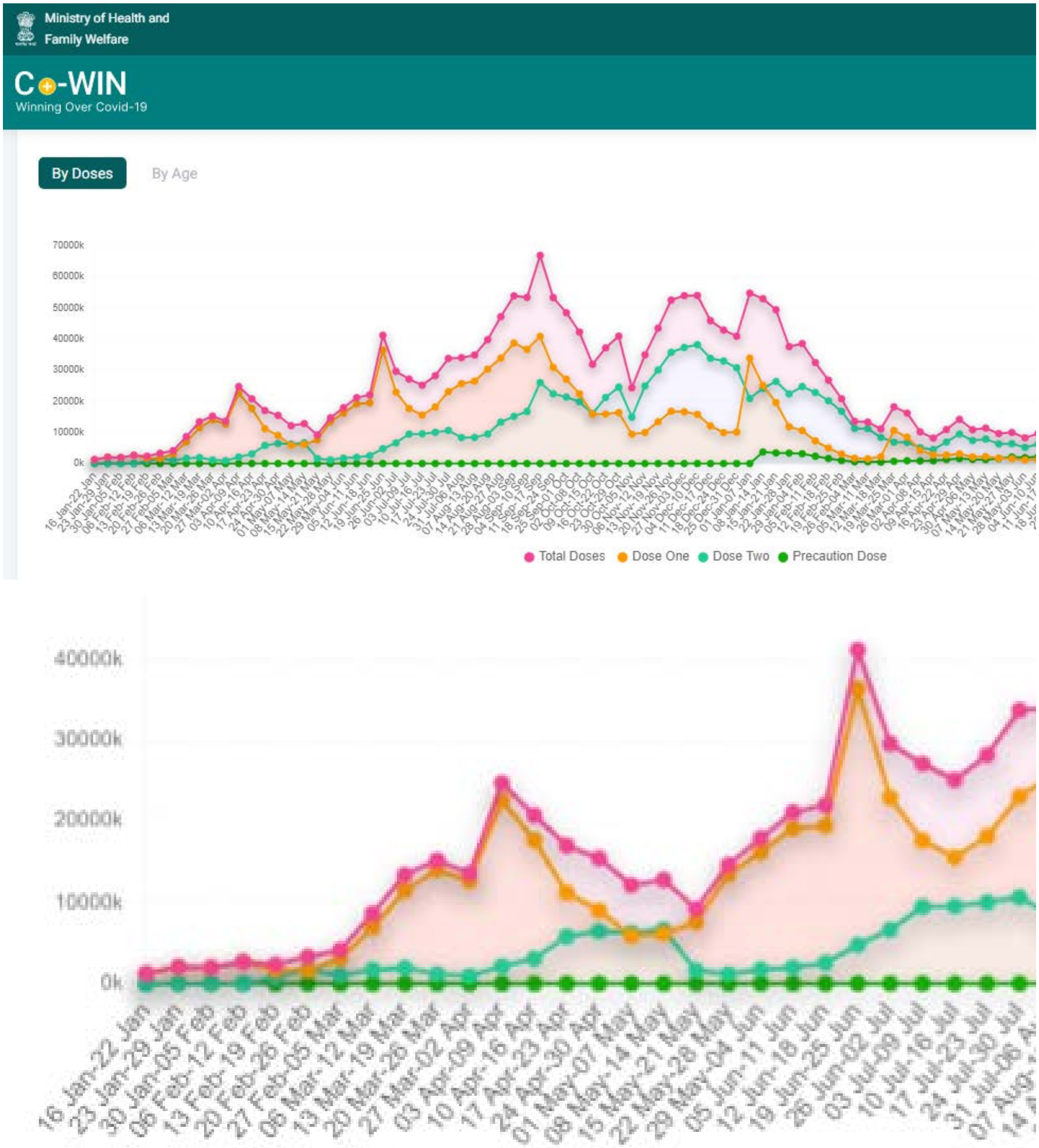


Figure 8: Ministry of Health and Family Welfare, Government of India (2022): C+ WIN dashboard, by dose type as indicated. (Upper) Broad view. (Lower) Selected enlargement.

For the following reasons (presented as numbered points), taken together, we conclude that the 4-month April-July 2021 surge in excess all-cause mortality in India may largely or predominantly have been caused by the vaccine rollout in its early stages.

1 - The mortality event is unique to India, sudden, unprecedented, massive and synchronous with India's vaccine rollout to the most elderly and most fragile (comorbidity) residents (Figures 1-8).

2 - By comparison, in relative terms, there were no significant mortality events and there was no significant cumulative excess mortality prior to April 2021, during more than a year of the declared pandemic (Figures 1-6).

The declared pandemic would have had to spare India for more than a year, while it raged in many other places around the world, before it showed a dramatic many-fold increase in virulence, suddenly in April-July 2021, when vaccines coincidentally were being rolled out to the elderly and those having comorbidities.

3 - The early rollout of the vaccine was not executed following the original ambitious plan but instead was at first delayed by implementation difficulties and then boosted by an *ad hoc* government intervention (Prime Minister Modi's 11-14 April 2021 *Teeka Utsav*, "Vaccine Festival"), which encouraged accelerated blanket and penetrating delivery to the poor, uneducated, and those presumed to be most in need.

4 - A similar synchronicity between increased vaccination associated with a government intervention to accelerate vaccine delivery and an anomalous surge (peak) in all-cause mortality is observed in connection with the so-called "vaccine equity" campaigns in the USA. An anomalous fall-2021 peak was interpreted as being caused by the vaccines, and is prominent in the 25-64 years age group in 21 states of the USA, most notably

including Alabama, Mississippi, Georgia, Florida and Louisiana (Rancourt et al., 2022). The data for Mississippi is shown below (Figure 9).

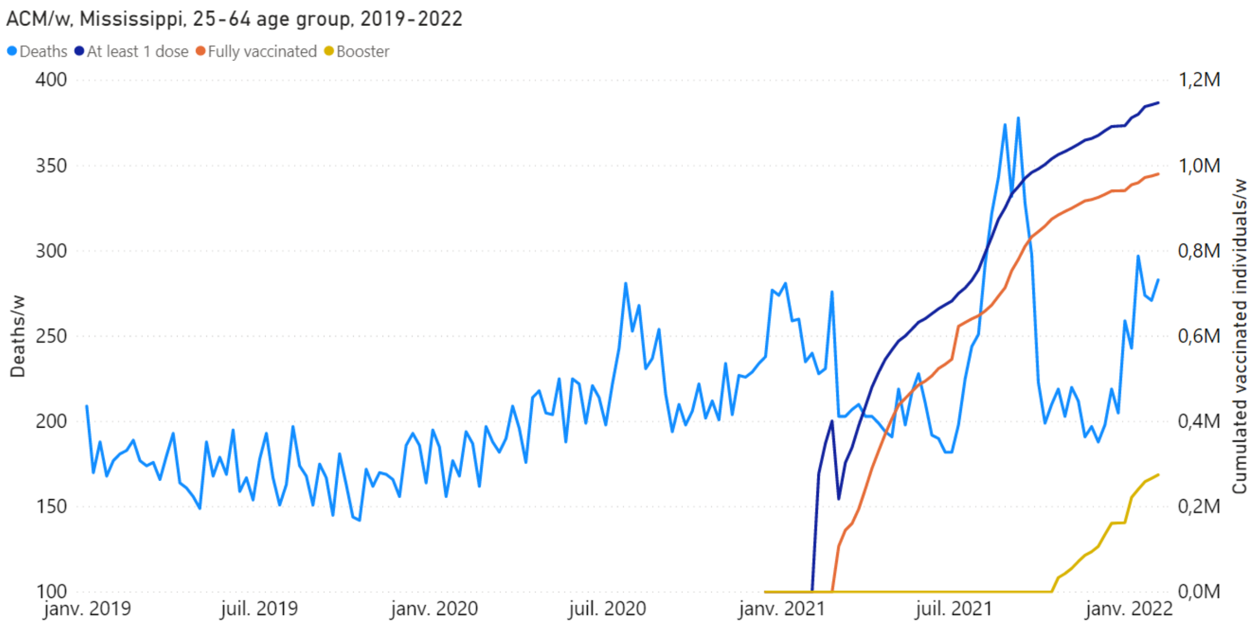


Figure 9: Rancourt et al. (2022), their Fig. 11B. All-cause mortality by week (light-blue), cumulated number of people with at least one dose of vaccine (dark-blue), cumulated number of fully vaccinated people (orange) and cumulated number of people with a booster dose (yellow) by week from 2019 to 2022, for 25-64 years age group in Mississippi. Data are displayed from week-1 of 2019 to week-5 of 2022.

In the study by Rancourt et al. (2022), it was concluded that significant (detectable by all-cause mortality) vaccine-induced mortality occurred primarily among fragile groups, characterized by high degrees of poverty, disability, obesity, diabetes, and high medication rates. The vaccine injection was seen as an additional challenge, often accelerating and causing death in residents with comorbidities.

5 - The magnitude of the April-July 2021 excess all-cause mortality event (normalized by population) is highly heterogeneous from region to region in India (above-cited references). This suggests that the net regional excess mortality is related to the underlying heterogeneity of health status, and to differences in health-status group selection, which were actually vaccinated in a region; rather than being due to a given

infection fatality ratio (and its age profile) for the rapid spread of an infectious disease, applied to all regions similarly.

6 - The April-July 2021 excess all-cause mortality event occurs simultaneously across India, as do the national vaccine rollout and Prime Minister Modi's "Vaccine Festival" intervention, rather than showing any distribution of starting times, which would be compatible with a spreading infectious disease seeding different regions at different times and spreading at different rates depending on regional differences of social and health conditions.

7 - The April-July 2021 excess all-cause mortality, at least initially, is significantly larger, on a mortality-baseline-percent basis for mortality by week, for 40-64 year old residents than for 65+ year old residents (~880% vs ~570%) (Figure 5). This is incompatible with controlled clinical studies and empirical observations, which find that infection fatality probability of COVID-19-assigned death is exponential with age (Bonanad et al., 2020; Goldstein and Lee, 2020; Santesmasses et al., 2020; Bauer et al., 2021; Elo et al., 2022; Sorensen et al., 2022). However, the age-dependence behaviour is similar to what is observed for the vaccination period of the Covid period compared to the pre-vaccination Covid period in the USA (Rancourt et al., 2022; see their Fig. 17).

8 - The Vaccine Adverse Event Reporting System (VAERS) of the USA unambiguously shows excess all-cause deaths immediately following injections with each of the three types of COVID-19 vaccines used in the USA, with a prominent peak within 5 days of injection and an exponentially decaying excess mortality extending 2 months following injection (Hickey and Rancourt, 2022; see their Figs. S3 through S5). The integrated mortality by number of injections following injection (injection toxicity) increases exponentially with age, as does the batch to batch variability of toxicity (Hickey and Rancourt, 2022; see their Fig. S6). The latter observations of exponential increases with age mean that the injections represent fatal challenges in proportion to frailty of the subject.

9 - Detailed histopathological and immunohistochemical autopsy studies have demonstrated that the COVID-19 vaccines are causes of death, both in otherwise healthy subjects and in elderly subjects with comorbidities (Choi et al., 2021; Schneider et al., 2021; Sessa et al., 2021; Gill et al., 2022; Mörz, 2022; Schwab et al., 2022).

10 - We have not found any study establishing that there was a sudden rise (and fall) of any disproportionately virulent variant of concern that would have been synchronous with or swept through and caused the April-July 2021 excess all-cause mortality event. For example, Dhar et al. (2021) postulate that the April-July 2021 “second wave” event in Delhi (the capital city of India) was due to the Delta variant, which would have quickly swept Delhi to become predominant because it would have higher transmissibility and larger immune escape than concomitantly circulating variants. However, Dhar et al. estimate the needed characteristics of Delta by fitting a model to the epidemiological data and to the variant predominance estimated by genomic measurements from small non-randomized cohorts. Leaving aside the large known and unknown uncertainties throughout their exercise, basically, the inferred characteristics of Delta are obtained by fitting to the data, rather than being independently measured in a controlled clinical trial. Under such circumstances, the mortality event creates an illusion of the needed Delta, but an actual Delta cannot be concluded to have caused the mortality event.

In conclusion, it appears that the early rollout of the vaccine in April-July 2021 in India was devastating, causing the deaths of approximately 3.7 million residents (Figure 1), on administering approximately 350 million doses of the vaccine (in a population of 1.39 billion).

This corresponds to an effective vaccine fatality per dose ratio (per-dose toxicity) of approximately 1%, which is approximately x100 the vaccine fatality per dose ratio for the Janssen vaccine administered to 65+ year old residents of the USA, calculated from the VAERS data (Hickey and Rancourt, 2021; see their Table 1). It is also

approximately the same vaccine fatality per dose ratio (1%) as is consistent with the anomalous fall-2021 peak in excess all-cause mortality occurring in high-poverty states of the USA, which was interpreted as being caused by the vaccine: Rancourt et al. (2022) and see the data for Mississippi shown in Figure 9.

Frail residents are susceptible to being fatally harmed on injection and should be protected against overly enthusiastic or politically motivated state-run injection campaigns implemented without stringent individual clinical risk assessment.

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Brief Report |
5 October 2022

Proof that Canada's COVID-19 mortality statistics are incorrect

Denis G. Rancourt[1,2,*], PhD

Marine Baudin[3], PhD

Jérémie Mercier[3], PhD

[1] Correlation Research in the Public Interest (correlation-canada.org)
[2] Ontario Civil Liberties Association (ocla.ca)
[3] Mercier Production (jeremie.mercier.org)
* denis.rancourt@alumni.utoronto.ca

This Brief Report is made public at the following websites.

https://correlation-canada.org/

https://ocla.ca/

https://denisrancourt.ca/

https://archive.today/

Abstract

We make a quantitative comparison between the COVID-19 mortality statistics of the Government of Canada (Public Health Agency of Canada; managed by the Chief Public Health Officer) and calculated total excess all-cause mortality (ACM) (deaths from all causes) for the Covid period. The claimed "COVID-19 deaths" mortality is almost double the total excess ACM for the same period, which we find to be irreconcilable with reality. We describe how these numbers have been uncritically used in public Government communications, by leading media, and in a recent scientific article co-authored by Canada's Chief Public Health Officer, which claims that "without the use of restrictive measures and without high levels of vaccination, Canada could have experienced […] almost a million deaths." We conclude that the COVID-19 mortality statistics are unreliable at best, and possibly meaningless.

Introduction

In Canada and in the world, there were virtually no reported deaths assigned to COVID-19 prior to the 11 March 2020 World Health Organization (WHO) declaration of a pandemic. Likewise, no anomaly in all-cause mortality by time (day, week, month) can be detected prior to the said declaration.[1]

The Government of Canada records "COVID-19 deaths" and reports the cumulative value on a weekly basis, at its Public Health Agency of Canada "COVID-19 epidemiology update" dashboard.[2]

[1] Rancourt, D.G. (2020) "All-cause mortality during COVID-19: No plague and a likely signature of mass homicide by government response", *ResearchGate*, 2 June 2020. https://doi.org/10.13140/RG.2.2.24350.77125 | archived at: https://archive.ph/PXhsg
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Government of Canada officers and employees use the same cumulative “COVID-19 deaths” data in their peer-reviewed scientific articles (see below).

This brief report is about the irreconcilable discrepancy between the Government of Canada’s numbers of “COVID-19 deaths” and rigorous evaluations of excess total all-cause mortality (ACM) for the same time periods.

What the Canadian Government and legacy media say

Table 1 presents statements made by the Government of Canada and by leading media, reporting cumulative “COVID-19 deaths”. The list is incomplete.

Table 1. COVID-19 death count statements

Statements by Canadian government and mainstream media regarding COVID-19 deaths		
#	**Statement**	**Source**
Government statements:		
1	“Table 1: **38,783** Deaths from coronavirus disease 2019 [COVID-19] Observed as of **April 24, 2022**.”	Ogden et al. (with Canada’s Chief Public Health Officer **Theresa Tam**), ***CCDR***, 2022.[3]
2	"COVID-19 cases deceased in Canada as of **September 23, 2022**, 7 am ET (n=**45,795** - This figure is based on cases for which a case report form was received by the Public Health Agency of Canada from provincial or territorial partners.)"	“COVID-19 epidemiology update”, **Government of Canada (Public Health Agency of Canada)**, Updated: September 23, 2022, 8 am ET.[4]

[3] Ogden NH, Turgeon P, Fazil A, Clark J, Gabriele-Rivet V, Tam T, Ng V. "Counterfactuals of effects of vaccination and public health measures on COVID-19 cases in Canada: What could have happened?" *Canada Communicable Disease Report* (CCDR) 2022;48(7/8):292–302. https://doi.org/10.14745/ccdr.v48i78a01

[4] https://health-infobase.canada.ca/src/data/covidLive/Epidemiological-summary-of-COVID-19-cases-in-Canada-Canada.ca.pdf (accessed on 27 September 2022).

3	"COVID-19 cases deceased in Canada as of **April 1, 2022**, 8 am EST (n=**36,992** - This figure is based on cases for which a case report form was received by the Public Health Agency of Canada from provincial or territorial partners.)	"COVID-19 epidemiology update", **Government of Canada (Public Health Agency of Canada)**, Updated: April 4, 2022, 9 am EST.[5]
Media statements:		
4	"At least 1 in 830 residents have died from the coronavirus, a total of **45,263** deaths." "Updated **Sept. 27, 2022**"	"Tracking Coronavirus in Canada: Latest Map and Case Count", ***New York Times***.[6]
5	"THE LATEST ON **SEPT. 23 [2022]** . Newly confirmed COVID-19 cases have brought the national total to over 4.23 million cases and more than **45,100** deaths."	"Coronavirus Tracker", ***Global News***.[7]
6	"A total of **16,409** people **[in Quebec]** have died from COVID-19 since the pandemic began." "Updated **Sept. 2, 2022** 2:26 p.m. EDT"	"COVID-19 hospitalizations down by 42 in Quebec", ***CTV News***.[8]
7	"More than **43,500** Canadians have died from COVID-19." "Thu., **Aug. 25, 2022**"	"Did a Conservative leadership hopeful compare COVID-19 vaccines to Nazi atrocities? Leslyn Lewis rejects 'cowardly' accusation", ***Toronto Star***.[9]
8	"Canada, meanwhile, has seen a total of **43,505** COVID-19-related deaths in the country since the pandemic began, including 251 people who died during the week of Aug. 7 to 13, according to the latest available data from Health Canada." "Posted **August 25, 2022** 12:53 pm"	"'Tragic milestone': 1M people have died of COVID-19 so far this year, WHO says", ***Global News***.[10]

[5] *Ibid.* (accessed after 4 April 2022)
[6] https://www.nytimes.com/interactive/2021/world/canada-covid-cases.html | Archived: https://archive.ph/puy6S (accessed on 27 September 2022).
[7] https://globalnews.ca/news/6649164/canada-coronavirus-cases/ (accessed on 27 September 2022).
[8] https://montreal.ctvnews.ca/covid-19-hospitalizations-down-by-42-in-quebec-1.6053545 (accessed on 27 September 2022).
[9] https://www.thestar.com/politics/federal/2022/08/25/did-a-conservative-leadership-hopeful-compare-covid-19-vaccines-to-nazi-atrocities-leslyn-lewis-rejects-cowardly-accusation.html | Archived: https://archive.ph/iTEjc (accessed on 27 September 2022).
[10] https://globalnews.ca/news/9084719/covid-deaths-hit-one-million-who/ (accessed on 27 September 2022).

9	“**43,583** deaths” “Last Updated Tuesday, **July 19, 2022** 12:15PM EDT”	“Tracking every case of COVID-19 in Canada”, ***CTV News***.[11]
10	“**42,254** coronavirus-related deaths reported in the country since the pandemic began.” “Last updated **July 15, 2022**”	“REUTERS COVID-19 TRACKER - Canada”, ***REUTERS***.[12]
11	“At least **41,000** Canadians (13,000 people in Ontario) have died from COVID-19 since the pandemic began. And, although we no longer see it as headline news, people are still dying every day from COVID-19 in our own cities and rural and remote areas.” “**May 30, 2022**”	“Kaplan-Myrth: Ontario election — COVID-19 isn't over. Vote for the party that will act on this reality”, ***Ottawa Citizen***.[13]
12	“Canada has reached another grim milestone: **40,000** COVID-19 deaths.” “PUBLISHED **MAY 13, 2022**”	“Canada reaches a grim milestone – 40,000 COVID-19 deaths”, ***The Globe and Mail***.[14]
13	“At least **40,000** people across Canada have died after contracting COVID-19 since the pandemic began more than two years ago, according to provincial data, and more than 70 people are still dying per day.” “Posted **May 13, 2022** 9:56 pm”	“Over 40,000 have died from COVID-19 in Canada, but hospitalizations are falling again”, ***Global News***.[15]
14	“The U.S. has experienced 302.93 deaths for every 100,000 people, per Johns Hopkins, a rate significantly higher than in Canada, with 104.30 deaths for every 100,000 people. Nearly **39,000** people have died in Canada.” “Posted: **May 12, 2022** 10:58 AM ET | Last Updated: May 12”	“U.S. surpasses 1 million COVID-19 deaths: A look at the numbers”, ***CBC News***.[16]

[11] https://www.ctvnews.ca/health/coronavirus/tracking-every-case-of-covid-19-in-canada-1.4852102 (accessed on 28 September 2022).
[12] https://graphics.reuters.com/world-coronavirus-tracker-and-maps/countries-and-territories/canada/ (accessed on 28 September 2022).
[13] https://ottawacitizen.com/opinion/kaplan-myrth-ontario-election-covid-19-isnt-over-vote-for-the-party-that-will-act-on-this-reality (accessed on 28 September 2022).
[14] https://www.theglobeandmail.com/canada/article-canada-40000-covid-19-deaths/ | Archived: https://archive.ph/v3w1r (accessed on 28 September 2022).
[15] https://globalnews.ca/news/8834765/covid-canada-40k-deaths-6th-wave/ (accessed on 29 September 2022).
[16] https://www.cbc.ca/news/world/us-million-covid-deaths-1.6150574 (accessed on 28 September 2022).

Clearly, these numbers are an integral part of the Government of Canada's communication campaign during the Covid period.

In addition, countless audio and video recorded interviews have media interviewers and commentators advancing these and comparable large cumulative numbers of "COVID-19 deaths", typically to emphasize the seriousness of the declared pandemic, and always implying that infection with the presumed SARS-CoV-2 virus was the dominant or only medical factor causing the deaths.

The detailed time evolution of the cumulative number of "COVID-19 deaths" is available at the Government of Canada (Public Health Agency of Canada) dashboard and its csv-file download,[17] and is represented in the following graph (Figure 1), in which the time axis starts on 1 February 2020.

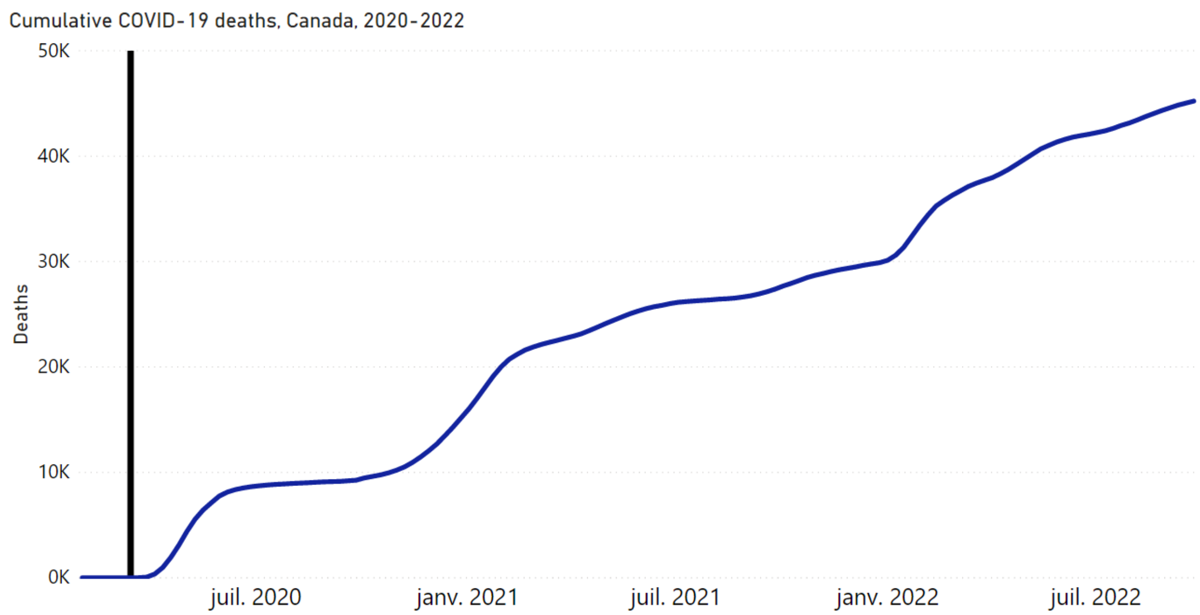


Figure 1. Time evolution of the cumulative number of "COVID-19 deaths" for Canada. The vertical line marks the week of 11 March 2020, when a pandemic was declared by the WHO. Data is from the Government of Canada (accessed on 3 October 2022).[18]

[17] See Footnote 2
[18] See Footnote 2

The same data as in Figure 1, viewed in terms of weekly new “COVID-19 deaths”, for the same time period (February 2020 to present), is shown in Figure 2.

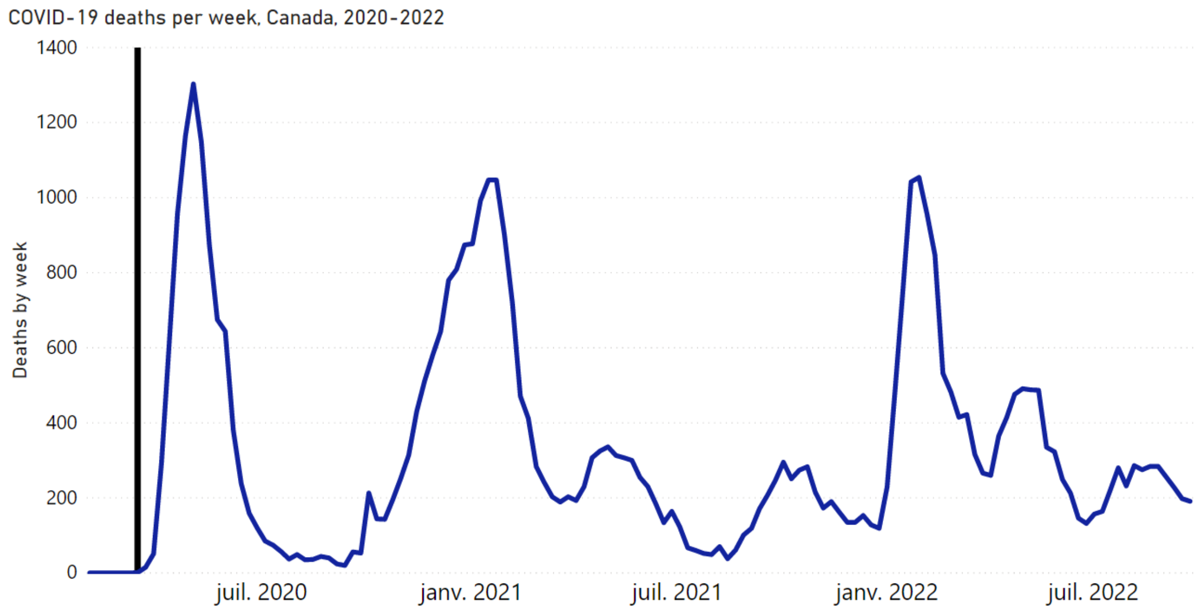


Figure 2. Time evolution of the weekly new number of “COVID-19 deaths” for Canada. The vertical line marks the week of 11 March 2020, when a pandemic was declared by the WHO. Data is from the Government of Canada (accessed on 3 October 2022).[19]

There is a consensus in the Government of Canada and the major media outlets that these numbers of “COVID-19 deaths”, reviewed above, represent true and reliable mortality caused by the SARS-CoV-2 virus, since COVID-19 is uniquely ascribed to this virus.

We were not able to find any Government of Canada sources or publications that suggested that the presumed virus could have played an insignificant or minor role in causing the deaths in some of the deaths attributed to or associated with “confirmed” COVID-19; nor were we able to find any Government (or investigative media) effort to estimate the fraction of any such “false positive” attributions of cause of death.

[19] See Footnote 2

What the all-cause mortality says

All-cause mortality by time is the most reliable data for detecting and epidemiologically characterizing events causing death, and for gauging the population-level impact of any surge or collapse in deaths from any cause. Such data is not susceptible to reporting bias or to any bias in attributing causes of death. More and more researchers are recognizing that it is essential to examine ACM by time, and excess deaths from all causes compared with projections from historic trends, to help make sense of the events surrounding COVID-19: See Rancourt et al. (2022)[20] and references therein.

Before we describe the quantification method, it is instructive to examine the ACM by time in Canada over the last three decades. Figure 3 shows ACM by month for Canada, from January 1991 through December 2020. Contrary to usual practice, we use the full y-scale, showing the zero, so that one may evaluate the relative importance of the seasonal variations and of any other changes compared to numbers of all the deaths in the country. This provides a reference to ascertain the degree to which the declared pandemic caused a notable excess in mortality after 11 March 2020.

[20] Rancourt, D.G., Baudin, M., Mercier, J. "COVID-Period Mass Vaccination Campaign and Public Health Disaster in the USA - From age/state-resolved all-cause mortality by time, age-resolved vaccine delivery by time, and socio-geo-economic data", *Research Gate*, 2 August 2022, DOI:10.13140/RG.2.2.12688.28164, https://www.researchgate.net/publication/362427136_COVID-Period_Mass_Vaccination_Campaign_and_Public_Health_Disaster_in_the_USA_From_agestate-resolved_all-cause_mortality_by_time_age-resolved_vaccine_delivery_by_time_and_socio-geo-economic_data | archived here: https://archive.ph/lFNwK

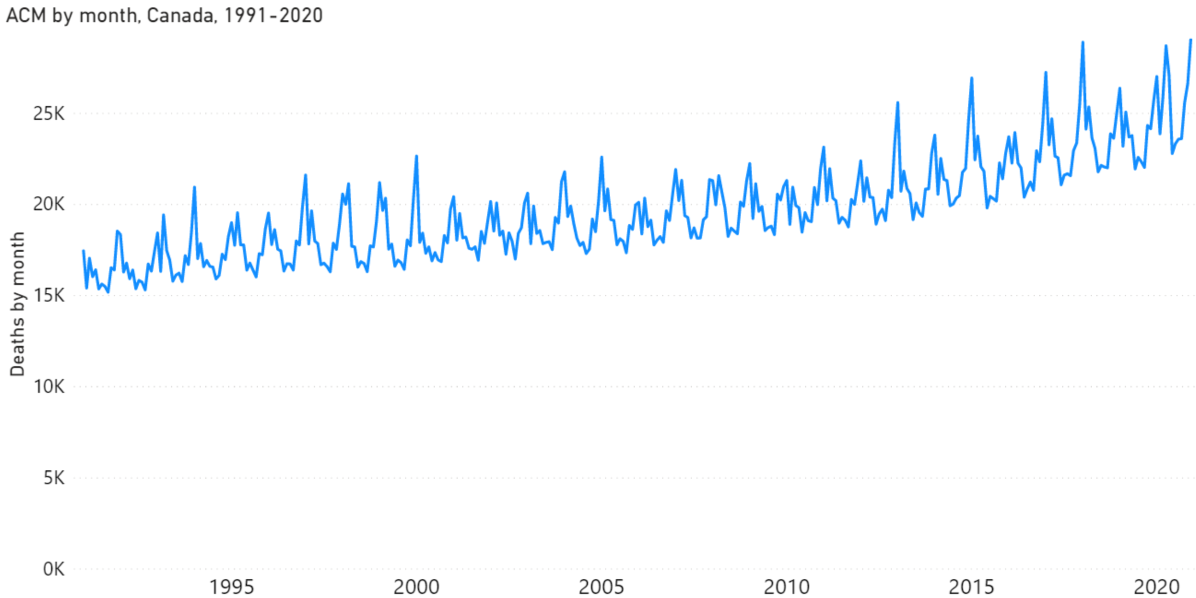


Figure 3. All-cause mortality (ACM) by month for Canada, from January 1991 to December 2020, inclusive. The data is from StatCan.[21] There are characteristic dips in February, due to the known artifact arising from February typically having only 28 days. The March-May 2020 peak that occurs immediately following the pandemic announcement of 11 March 2020 is historically anomalous, and we have discussed it previously.[22]

Next, we apply similar quantitative methods that we applied recently for the USA[23] to the case of Canada, to quantify excess total ACM for the Covid period, which started on 11 March 2020. By "excess" we mean in addition to the expected mortality for the Covid period, based on the historic trend prior to 11 March 2020. As such, the expected mortality for the Covid period is the mortality that one would predict if the Covid period were just like recent prior periods, in terms of the factors that determine mortality.

[21] StatCan (2022) "Deaths, by month". Release date: 2022-01-24. https://www150.statcan.gc.ca/t1/tbl1/en/tv.action?pid=1310070801 (accessed on 6 June 2022).
[22] Rancourt, D.G., Baudin, M. and Mercier, J. (2021) "Analysis of all-cause mortality by week in Canada 2010-2021, by province, age and sex: There was no COVID-19 pandemic and there is strong evidence of response-caused deaths in the most elderly and in young males". *ResearchGate*, 6 August 2021, https://doi.org/10.13140/RG.2.2.14929.45921 | archived here: https://archive.ph/CYA20
[23] Rancourt et al. (2022): *Footnote 20.*

We use the StatCan data of ACM by week,[24] which starts at the week ending Saturday 9 January 2010, and ends at the week ending Saturday 14 May 2022. Although StatCan refers to this data as “provisional weekly death counts”, we have observed that successive updates for this product (their Table 13-10-0768-01) do not change the previously released data to a degree that could significantly change our calculations or conclusions. The last values in the dataset for May do not appear to be anomalous.

Given the end date of the data and given the start date of 11 March 2020 of the declared pandemic, the Covid period used in our calculation (the “defined Covid period”) is the 114-week period between the week ending Saturday 14 March 2020 and the week ending Saturday 14 May 2022, inclusive. We sum ACM over this 114-week period. We define non-overlapping 114-week periods of summation of ACM, which immediately precede the defined Covid period. Four such consecutive periods prior to the defined Covid period can be accommodated by the data.

We plot the resulting sum of ACM values versus time, along with the ACM by week (on a different y-axis), in Figure 4.

[24] StatCan (2022) “Table 13-10-0768-01 Provisional weekly death counts, by age group and sex”. Release date: 2022-09-08. https://doi.org/10.25318/1310076801-eng | also: https://www150.statcan.gc.ca/t1/tbl1/en/tv.action?pid=1310076801 (accessed on 12 September 2022)

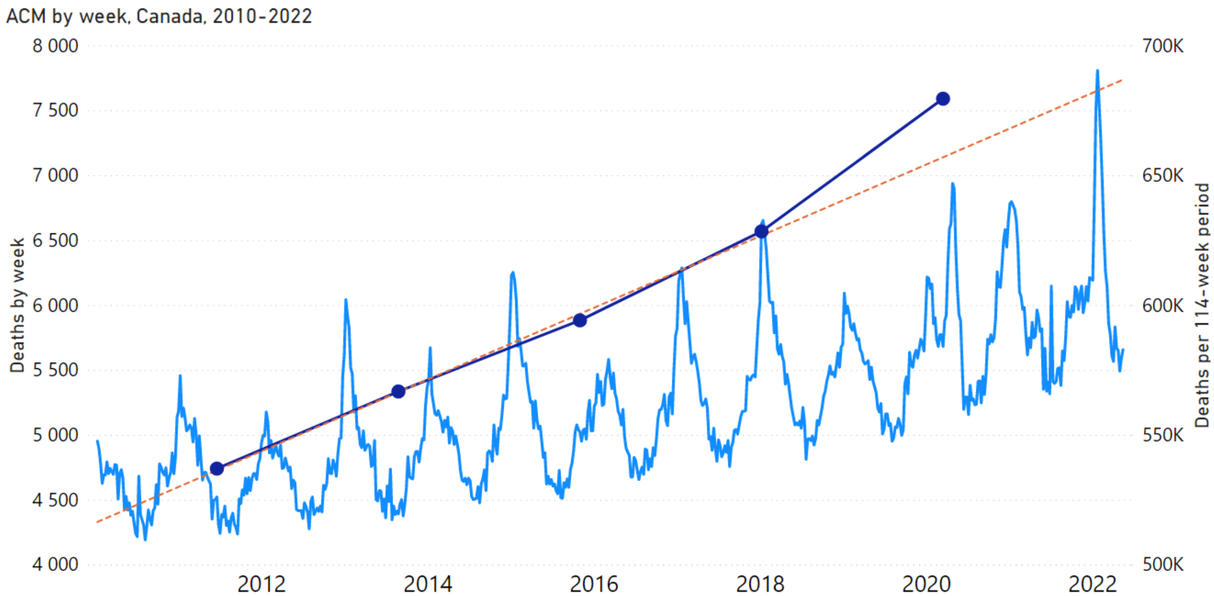


Figure 4. All-cause mortality (ACM) by week, 2010-2022, left y-axis (light blue continuous curve) for Canada; and ACM sums over the five 114-week non-overlapping consecutive periods described in the text, right y-axis (dark blue dots, joined by line segments). The ACM sums are positioned in time on the x-axis at the first week of the respective summation period. The last 114-week period is our operational Covid period (the defined Covid period). The orange straight dashed line is the least-squares best fit to the four ACM sums prior to the defined Covid period. The sharp spike occurring in the summer of 2021 corresponds to the heat wave that occurred in British Columbia (and the north-western USA).

We make a least-squares fit of a straight line to the four ACM sums of the 114-week periods prior to the defined Covid period (shown in Figure 4). Taking “x” to be the week number, where x=1 is the first week in the StatCan data, the resulting fitted line has slope = 264.5 ACM-sum-on-114-weeks per week, intercept = 516,400 deaths in 114-week period, and Pearson correlation coefficient *r* = +0.9989.

Therefore, the expected 114-week ACM sum for the defined Covid period, based on the least-squares fitted straight line, is (657.1 ± 1.3) x 10^3 deaths, where the uncertainty is estimated as the mean of the four absolute values of the deviations of the observed values from the fitted line; whereas the measured ACM sum for the 114-week defined Covid period is 679,645 deaths.

This means that the excess mortality for the 114-week defined Covid period ending on the week ending on Saturday 14 May 2022, is:

$$679{,}645 \; - \; (657.1 \pm 1.3) \times 10^3 \; = \; \mathbf{(22.5 \pm 1.3) \times 10^3} \text{ deaths,}$$

which is seen in Figure 4.

Covid-assigned deaths versus all-cause mortality

The thus obtained excess ACM for the 114-week defined Covid period ending on 14 May 2022 can be compared to the cumulative “COVID-19 deaths” on 14 May 2022.

The latter official value for 14 May 2022, from the Government of Canada (Public Health Agency of Canada), is: **40,684** “COVID-19 deaths”.[25]

Therefore, we have:

40,684 “COVID-19 deaths” (up to 14 May 2022) vs 22,500 ± 1,300 total excess all-cause deaths (up to 14 May 2022)

This means that there were 18,200 more “COVID-19 deaths” than the 22,500 excess all-cause deaths (up to 14 May 2022).

[25] See Footnote 2

The "COVID-19 deaths" mortality, in magnitude, is 181% of the calculated total excess ACM (up to 14 May 2022).

If the same ratio were applied to the USA, there would have been 1.81 x 1.27M[26] = 2.30M "COVID-19 deaths" in the USA, more than double the official USA number (998,587 "COVID-19 Deaths" on 14 May 2022, CDC).[27]

It is inconceivable that a virus killed this many more people than the total excess ACM, because this would imply that in the absence of the presumed virus there would be a large deficit of ACM. Alternatively, one would need to believe that Covid measures (masking, social distancing, isolation, shutting down economic sectors, etc.) cause a net reduction of deaths from all other causes; such as not causing any deaths while more than eliminating "influenza and pneumonia", which in Canada have reported deaths in the range 6.2 to 8.6 K/year for 2016 through 2019.[28]

The presumed SARS-CoV-2 virus would have killed approximately twice as many people as the calculated excess ACM. This means that, in addition to presumably being the cause for all the excess ACM (which is implausible), the presumed SARS-CoV-2 virus would have also had to rush in and kill 18,200 people, in the same time period and before they could die of other causes, who most certainly would have died without the Covid circumstances. What is the meaning of a presumed virulent virus that kills people who would have died, when they would have died? Alternatively, for example, the Covid measures would have saved 18,200 people from "influenza and pneumonia", say, while the presumed SARS-CoV-2 virus killed them.

More realistically, if approximately half of the excess deaths were due to the aggressive measures (including: harmful medical treatment, neglect of vulnerable individuals, social

[26] Rancourt et al. (2022): Footnote 20.
[27] "COVID Data Tracker - Trends in Number of COVID-19 Cases and Deaths in the US Reported to CDC, by State/Territory", CDC, https://covid.cdc.gov/covid-data-tracker/#trends_totaldeaths_select_00 (accessed on 2 October 2022).
[28] "Leading causes of death, total population, by age group", Table: 13-10-0394-01 (formerly CANSIM 102-0561), Release date: 2022-01-24, Statistics Canada, https://www150.statcan.gc.ca/t1/tbl1/en/tv.action?pid=1310039401 (accessed on 2 October 2022).

and physical isolation, and loss of regular occupation and care protocols), then at most 10,000 or so deaths could have been caused by the presumed SARS-CoV-2 virus, in this period, and the reported number of "COVID-19 deaths" is inflated by a factor of approximately 4, if the cause-of-death determinations can be taken to be meaningful.

Discussion: What does the Government of Canada say?

Deputy Prime Minister of Canada Chrystia Freeland[29] has stated that if Canada had the same "COVID-19 deaths" rate per capita as the USA, then there would have been 70,000 more COVID-19 deaths in Canada.[30] Freeland referred to a study by Naylor and other academics as her source. Razak et al. (including Naylor) make their analysis up to or near 12 February 2022 when the reported cumulative "COVID-19 deaths" for Canada were at 35,268. For this date, they report "COVID-19 deaths" rates per capita (per million) of 919 for Canada and 2,730 for the USA (their Figure 1C).[31] The USA rate would produce 105,000 deaths in Canada, which is 70,000 more than 35,000.

This statement by Freeland has a "COVID-19 deaths" rate for the USA, which is 3.0 times larger than for Canada, but Freeland does not mention two important factors:

(1) the USA has an excess-ACM death rate (per capita) that is 6.5 times larger than for Canada [(1.27M/22.5K)(38M/330M) = 6.5], and
(2) the Covid-measures stringency index (Oxford Stringency Index) is statistically indistinguishable for the USA and Canada [Figure 2 in Razak et al.[32]].

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[29] https://deputypm.canada.ca/en | archived: https://archive.ph/uyAHz (accessed on 1 October 2022).
[30] Video: "All-cause deaths continue to skyrocket in Canada", *Rebel News*, 26 September 2022. https://rumble.com/v1lmo2p-all-cause-deaths-continue-to-skyrocket-in-canada.html (at 4:12).
[31] Fahad Razak, Saeha Shin, C. David Naylor, Arthur S. Slutsky. "Canada's response to the initial 2 years of the COVID-19 pandemic: a comparison with peer countries" *CMAJ* Jun 2022, 194 (25) E870-E877; DOI: https://doi.org/10.1503/cmaj.220316 . See also the 27 June 2022 *Globe&Mail* opinion piece by Razak, Slutsky and Naylor: https://www.theglobeandmail.com/opinion/article-we-need-new-strategies-to-tackle-covid-this-fall/ | archived: https://archive.ph/moeYs .
[32] *Ibid.*

Freeland's attention should have been turned instead to a metric that takes into account the different health statuses of the vulnerable populations in the two countries.[33] Freeland could have asked herself: "Why is the ratio of 'COVID-19 deaths' to excess ACM deaths [(40.7K/22.5K)/(0.999M/1.27M)] some 2.3 times larger in Canada than in the USA?" This contextualized comparison would mean a relative (compared to the USA) catastrophic failure of the Covid measures intended to prevent spread of the disease in Canada, in which the presumed infection appears to have disproportionately devastated those close to death in Canada. Freeland misled herself in her use of the USA regarding comparative efficacy of Covid measures in Canada.

Discussion: What do the Government scientists say?

Ogden et al. (with Canada's Chief Public Health Officer Theresa Tam), publishing in the peer-reviewed journal *Canada Communicable Disease Report* (CCDR) in July/August 2022 wrote:[34]

> "Together, these observations show that without the use of restrictive measures and without high levels of vaccination, Canada could have experienced substantially higher numbers of infections and hospitalizations and almost a million deaths."

One million added "COVID-19 deaths" in Canada corresponds to adding approximately 150% of the baseline total (not excess) ACM deaths for the Covid period. This would increase the Covid-period total (not excess) ACM from approximately 680,000 deaths (Figure 4) to approximately 1,680,000 deaths. One can gauge what that would look like on Figures 3 and 4.

[33] Rancourt et al. (2022): Footnote 20.
[34] Ogden et al. (2022): Footnote 3.

To make it more visual and concrete, we simulate the ACM by week for Canada with the added said "almost a million deaths" in Figure 5. Here, for the sake of illustration and simplicity, we add the one million deaths to the defined Covid period uniformly to each of the 114 weeks in the period (1M/114 = 8,772 deaths added to each week in the defined Covid period; keeping in mind that the Ogden et al. article uses data up to 20 April 2022, which is close to our defined Covid period end date).

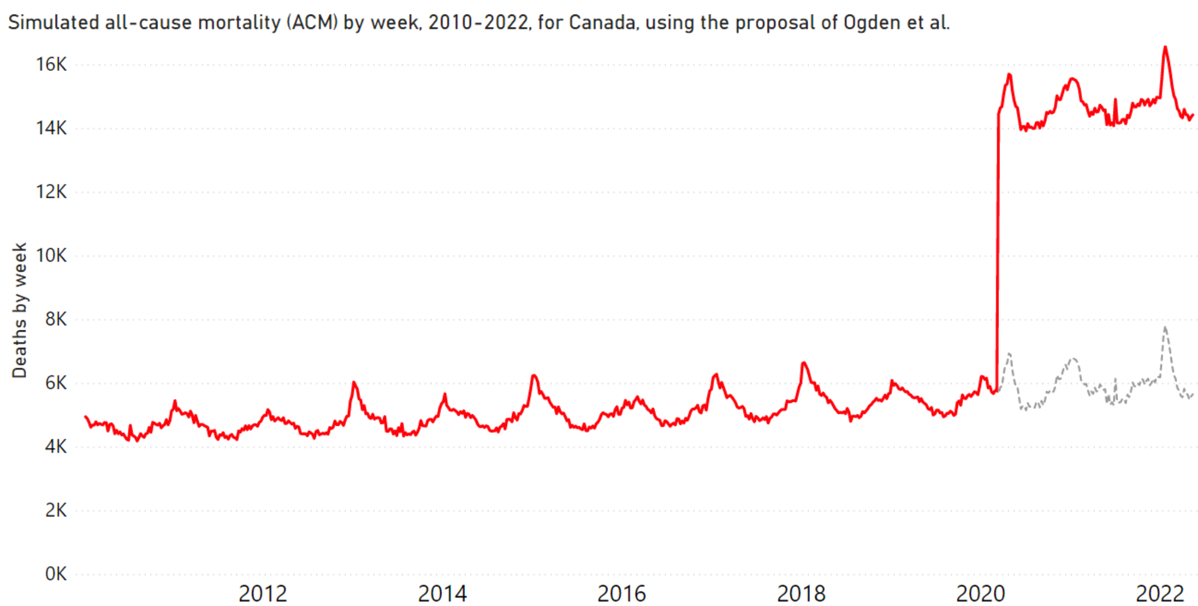


Figure 5. Simulated all-cause mortality (ACM) by week, 2010-2022, for Canada, using the proposal of Ogden et al. (red line), as explained in the text. The original data for the Covid period is shown by the dashed grey line.

Figure 5 suggests that the proposal made by Ogden et al. is not compatible with any reasonable view.

The theoretical notion that one million deaths were averted by the Covid measures in Canada is incredible on its face, but also contrary to reality. It would correspond to

210 million deaths globally [(1M/38M) x 8B]; and to 8.7 million deaths in the USA [(1M/38M) x 330M].

This conclusion by Ogden et al. (including Canada's Chief Public Health Officer Theresa Tam) is not connected to reality because, in addition to relying on reported "COVID-19 deaths" numbers, it is a product of their theoretical modelling exercise. All such models applied to nations have been shown to often be grossly unreliable. Arguably the most renowned epidemiologist (cited >450K times),[35] Stanford University's Professor of Medicine John Ioannidis and co-authors had this to say about the models:[36]

> "Epidemic forecasting has a dubious track-record, and its failures became more prominent with COVID-19. Poor data input, wrong modeling assumptions, high sensitivity of estimates, lack of incorporation of epidemiological features, poor past evidence on effects of available interventions, lack of transparency, errors, lack of determinacy, consideration of only one or a few dimensions of the problem at hand, lack of expertise in crucial disciplines, groupthink and bandwagon effects, and selective reporting are some of the causes of these failures. Nevertheless, epidemic forecasting is unlikely to be abandoned."

At this point, readers have a choice of preferring to side more with one of two end-point views. Either:

(a) the Government of Canada saved one million lives, and thereby brought down mortality coincidentally to virtually the same level as in the pre-Covid periods (Figures 3 and 4); within 22,500 deaths, which is approximately +3% of expected mortality in the absence of Covid circumstances; or

[35] Google Scholar authenticated profile of John P.A. Ioannidis: https://scholar.google.com/citations?user=JiiMY_wAAAAJ&hl (accessed on 1 October 2022).

[36] Ioannidis JPA, Cripps S, Tanner MA. "Forecasting for COVID-19 has failed". *Int J Forecast.* 2022 Apr-Jun;38(2):423-438. doi: 10.1016/j.ijforecast.2020.08.004. Epub 2020 Aug 25. PMID: 32863495; PMCID: PMC7447267. https://doi.org/10.1016/j.ijforecast.2020.08.004

(b) there was no such contagious and virulent pathogen present, and, if anything, the Covid measures may have caused net harm.

In making this evaluation, readers should keep in mind that the article by Ogden et al. (including Canada's Chief Public Health Officer Theresa Tam) is written by the architects of the Covid measures in Canada, and of the COVID-19 testing and vaccination campaigns. It is published by the Government. And it constructs a theoretical justification for unprecedented harsh nation-wide Government measures. It cannot be viewed as unbiased.

Conclusion

We determined the expected defined Covid period mortality (nominally from 11 March 2020 to 14 May 2022), in the absence of the Covid period circumstances to be: $(657.1 \pm 1.3) \times 10^3$ deaths.

The actual defined Covid period mortality was 679,645 deaths.

Therefore, the defined Covid period excess mortality is $(22.5 \pm 1.3) \times 10^3$ deaths, which is significantly smaller than the Government's reported "COVID-19 deaths" number of 40,684 for the same period.

These numbers (22.5K vs 40.7K) cannot be reconciled by any reasonable explanation, which we have explored.

The recent suggestion by Ogden et al., derived from using the Government-reported "COVID-19 deaths" mortality, that "without the use of restrictive measures and without high levels of vaccination, Canada could have experienced […] almost a million deaths.", appears to be palpably disconnected from reality (Figure 5).

In conclusion, our analysis overall leads us to suggest that the COVID-19 mortality statistics collected and presented by the Government of Canada (Public Health Agency of Canada) are unreliable at best, and possibly meaningless.

Compartmental mixing models for vaccination-status-based segregation regarding viral respiratory diseases

Joseph Hickey, PhD[1,*] and Denis G. Rancourt, PhD[1]

[1] Correlation Research in the Public Interest (correlation-canada.org)

*Corresponding author: joseph.hickey@ucalgary.ca

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Abstract

Background: Segregation of unvaccinated people from public spaces has been a novel and controversial COVID-era public health practice in many countries. Models can be used to explore potential consequences of vaccination-status-based segregation. The models must be simple enough to provide reliable predictions of possibilities, while including the essential ingredients to make them sufficiently realistic. We systematically investigate implementing effects of segregation on person-to-person contact frequencies and show this critically determines the predicted epidemiological outcomes.

Methods: We describe a susceptible-infectious-recovered (SIR) two-population model for vaccinated and unvaccinated groups of individuals that transmit an infectious disease by person-to-person contact. The degree of segregation between the two groups, ranging from zero to complete segregation, is implemented using the like-to-like mixing approach developed by Garnett and Anderson (1996) for sexually-transmitted diseases, adapted for presumed SARS-CoV-2 transmission. We allow the contact frequencies for individuals in the two groups to be different and depend, with variable strength, on the degree of segregation.

Results: Model predictions for a broad range of model assumptions and respiratory-disease epidemiological parameters are calculated to examine the predicted effects of segregation. Segregation can either increase or decrease the attack rate among the vaccinated, depending on the type of segregation (isolating or compounding), and the contagiousness of the disease. For diseases with low contagiousness, segregation can cause an attack rate in the vaccinated, which does not occur without segregation.

Interpretation: There is no blanket epidemiological advantage to segregation, either for the vaccinated or the unvaccinated. Negative epidemiological consequences can occur for both groups.

Introduction

Models can be used to investigate infectious disease dynamics under different hypotheses about the characteristics of a disease and the effects of health policy. In such applications, it is crucial to base the model on the simplest-possible sufficiently realistic conceptual foundation and only add extensions incrementally (Garnett & Anderson, 1996; Siegenfeld et al., 2020). This optimizes relevance and minimizes confounding the results with complexity and intangible propagation of error. Following this approach, researchers have extended the foundational SIR-type model to explore diseases with birth and death dynamics, maternal- or vaccine-derived immunity, latency of infection, and so on (Hethcote, 2000; Keeling & Rohani, 2008; Martcheva, 2015).

Recently, simple susceptible-infectious-recovered (SIR) models of epidemic dynamics have been implemented with two interacting societal groups (vaccinated and unvaccinated) to examine epidemic outcomes for variable degrees of interaction between the two groups, including whether the unvaccinated put the vaccinated unduly or disproportionately at risk, using epidemiological parameters presumed to be representative of SARS-CoV-2 (Fisman et al., 2022; Virk, 2022; Kosinski, 2021).

These prior implementations take the person-to-person contact frequencies of the majority and socially-excluded groups to be equal and held constant, irrespective of the degree of segregation (or exclusion or “like-to-like mixing”). In other words, in the previous models, the total number of contacts that an individual from either of the two societal groups experiences per day is constant and unaffected by the degree of segregation between the two groups.

Here, we implement person-to-person contact frequencies that can be different for the two groups and that vary with the degree of segregation, in different ways. We explore different modes and amplitudes of the variations of frequencies with degree of segregation, and their consequences on the predicted epidemiological outcomes. This is necessary because, for example, in many actual regulatory policies the excluded unvaccinated group is barred from public venues or services where people gather and from public transport where people are in close proximity for various durations.

In general, the person-to-person contact frequency of the excluded group decreases with increasing segregation if isolation is in effect, and increases with increasing segregation if the excluded individuals are in-effect put into compounds or camps. Implementing these essential model features gives rise to a rich and more complex epidemiological behaviour, whatever epidemiological parameters are used.

The Model

We adopt the standard SIR framework with two sub-populations, as has been done with sexually-transmitted diseases and was recently done with vaccination status.

Following the usual SIR model structure, a person can be in one of three states: susceptible to infection (S), infectious (I), or recovered and immune (R). If a susceptible person comes into contact with an infectious person, the susceptible person can become infectious, and infectious people eventually recover.

Our model population is divided into two groups: vaccinated and unvaccinated. Vaccination is "all or nothing", such that a proportion VE of the vaccinated population is immune (are in the R state from the outset of the simulation), where the parameter VE represents vaccine efficacy. The model also includes a natural immunity parameter, NI, equal to the proportion of unvaccinated that are immune from the outset.

The model parameter η controls the degree of segregation between vaccinated and unvaccinated people. When $\eta = 0$, there is no segregation, and the two groups mix randomly. When $\eta = 1$, there is complete segregation, such that vaccinated only come into contact with other vaccinated, and unvaccinated only come into contact with other unvaccinated.

The parameter η follows from Garnett and Anderson (1996), who modeled sexually-transmitted disease spread in a population divided into groups with different frequencies of sexual contacts. Since it is reasonable to assume that the level of desire for sexual contact is an intrinsic characteristic of individuals, it is reasonable to assume that segregation does not change the contact frequencies in either group in Garnett and Anderson's model. However, contact frequency is not generally and solely an intrinsic individual characteristic, and segregation based on vaccination status may increase or decrease contact frequencies, depending on how the segregation is implemented.

In our model, the contact frequencies of either vaccinated or unvaccinated individuals (or both) can increase, decrease, or remain constant as the two groups are segregated. This is controlled by the parameters m_v and m_u, which determine the degree of increase or decrease of the contact frequency in either group, as η is varied.

For example, when $m_u < 0$, as segregation is increased, the contact frequency of unvaccinated people, c_u, decreases. This corresponds to segregation policy that excludes unvaccinated people from public spaces, e.g., using vaccination passports. Conversely, when $m_u > 0$, then as segregation is increased, c_u increases. This corresponds to segregation policy that compounds unvaccinated people, for example in prisons or camps.

In principle, the vaccinated and unvaccinated contact frequencies may be different even when the two groups are completely unsegregated. The unsegregated ($\eta = 0$) contact frequencies are set by the parameters c_v^0 and c_u^0. Similarly, the probability that contact between a susceptible and infectious person results in transmission is β_v (β_u) for a susceptible vaccinated (unvaccinated) person and the rates of recovery from infection for the vaccinated and unvaccinated individuals are γ_v and γ_u, respectively.

There are thus two "β parameters", two "*c* parameters" and two "γ parameters" in our model. Since each β parameter always occurs as part of a product with its respective *c* parameter, the β parameters can freely be set equal to 1: this imposes that the "contacts" considered in the model are, by definition, only those contacts that are of sufficiently close proximity and long duration that an infection is guaranteed to occur when a susceptible and an infectious person meet. We set $\beta_v = \beta_u = 1$ in this paper, without any loss of generality. For a more contagious virus, more of an individual's contacts are long and close enough that transmission would be guaranteed, corresponding to higher c_v^0 and c_u^0.

The model of Fisman et al. (2022) is the special case of our model with $m_u = m_v = 0$, $c_v^0\beta_v = c_u^0\beta_u$ and $\gamma_v = \gamma_u$. When $m_u = m_v = 0$, the contact frequencies of both vaccinated and unvaccinated remain constant regardless of the level of segregation. Segregation then implies two parallel societies, where each population has its own public spaces (such as restaurants, airplanes, etc.) and within these spaces maintain the same contact frequencies they would have with no segregation. Fisman et al.'s implementation does not represent how segregation has been applied during the COVID era in Canada and many countries (Looi, 2021; Lawson et al., 2022), since unvaccinated people were excluded from public spaces while vaccinated people were allowed access.

We do not use the "basic reproduction number", R_0, since it would be derived from the fundamental parameters of the model. In a model with multiple sub-populations, the dynamics are not characterized by a single R_0 because the infection probabilities (β parameters), contact frequencies (*c* parameters) and recovery rates (γ parameters) are different for each sub-population, in general.

The parameters of our model are listed in Table 1; calculated quantities in Table 2. Technical details of the model are in Appendix 1.

Table 1: Model parameters

Parameter description	Symbol	Typical value	Bound
Degree of segregation between vaccinated and unvaccinated groups	η	(varied)	0 to 1
Probability of transmission per contact between a susceptible vaccinated person and an infectious person	β_v	1	0 to 1
Probability of transmission per contact between a susceptible unvaccinated person and an infectious person	β_u	1	0 to 1
Contact frequency of vaccinated people when η=0	c_v^0	438 contacts/yr	≥ 0
Contact frequency of unvaccinated people when η=0	c_u^0	438 contacts/yr	≥ 0
Degree of increase ($m_v > 0$) or decrease ($m_v < 0$) of vaccinated contact frequency as a function of η	m_v	0	≥ -1
Degree of increase ($m_u > 0$) or decrease ($m_u < 0$) of unvaccinated contact frequency as a function of η	m_u	(varied)	≥ -1
Rate of recovery of a vaccinated person (per year)	γ_v	73 yr^{-1}	≥ 0

Rate of recovery of an unvaccinated person (per year)	γ_u	73 yr^{-1}	≥ 0
Population fraction of vaccinated people	P_v	0.8	0 to 1
Vaccine efficacy	VE	0.2	0 to 1
Proportion of unvaccinated population with natural immunity	NI	0.2	0 to 1
Population of entire society	N	10^7	> 0

Table 2: Quantities calculated from model results (mathematical definitions in Appendix 1, Section A1.3)

Name	Symbol
Attack rate in the vaccinated population	A_v
Attack rate in the unvaccinated population	A_u
Share of infections among vaccinated people that were due to contacts with infectious unvaccinated people	B_v

Results

The attack rate among the vaccinated population is defined as the proportion of initially-susceptible vaccinated people who become infected during the epidemic: $A_v = \left(S_v(t_0) - S_v(t_f)\right)/S_v(t_0)$, where $S_v(t_0)$ is the number of susceptible vaccinated people at the beginning of the epidemic and $S_v(t_f)$ is the number of susceptible vaccinated people remaining once there are no longer any infectious people in the entire (vaccinated and unvaccinated) population. A_u is defined equivalently, for the unvaccinated.

We also define B_v as the share of infections among vaccinated people that were due to contacts with infectious unvaccinated people.

We focus on segregation types that are targeted at the unvaccinated group. We assume, for simplicity, that segregation has no impact on the contact frequency of vaccinated people (m_v = 0). We also assume that the contact frequencies in both groups are the same when there is no segregation ($c_v^0 = c_u^0$). We use the same values as used by Fisman et al. (2022) for the remaining parameters: P_v = 0.8, VE=0.8, NI=0.2, $\gamma_v = \gamma_u = 73$ yr^{-1}, and $N = 10^7$. These values were presumed to be representative for COVID-19 and vaccination.

Appendix 2 contains supplementary figures with results for different parameter combinations, including $m_v \neq 0$ and $c_v^0 \neq c_u^0$. In all results in this paper, simulations were initiated with a seed number of 100 infectious individuals distributed proportionately among the two sub-populations.

Fig. 1 shows results for a moderate value of $c_v^0 = c_u^0 = 300$. For reference, in a single-population model, c = 300, β = 1 and γ = 73 corresponds to R_0 = 4.1.

As can be seen in Fig. 1a, when $m_u < 0$ (exclusion and isolation of unvaccinated people) the vaccinated attack rate, A_v decreases with increasing segregation. However, when $m_u > 0$

(compounding of unvaccinated people), there is a maximum in A_v for moderate values of η. Therefore, with compounding segregation, very large values of η are required for A_v to be lower than its value with no segregation (η = 0). Fig. 1b is the unvaccinated attack rate, A_u versus degree of segregation, η. Fig. 1c shows that B_v, the share of vaccinated infections that are due to unvaccinated people, has a shape similar to $A_v(\eta, m_u)$. In all panels, 20% of the total population is unvaccinated (P_v = 0.8; Table 1).

Fig. 1 therefore demonstrates that whether segregation increases or decreases the vaccinated-population attack rate depends on how segregation is implemented.

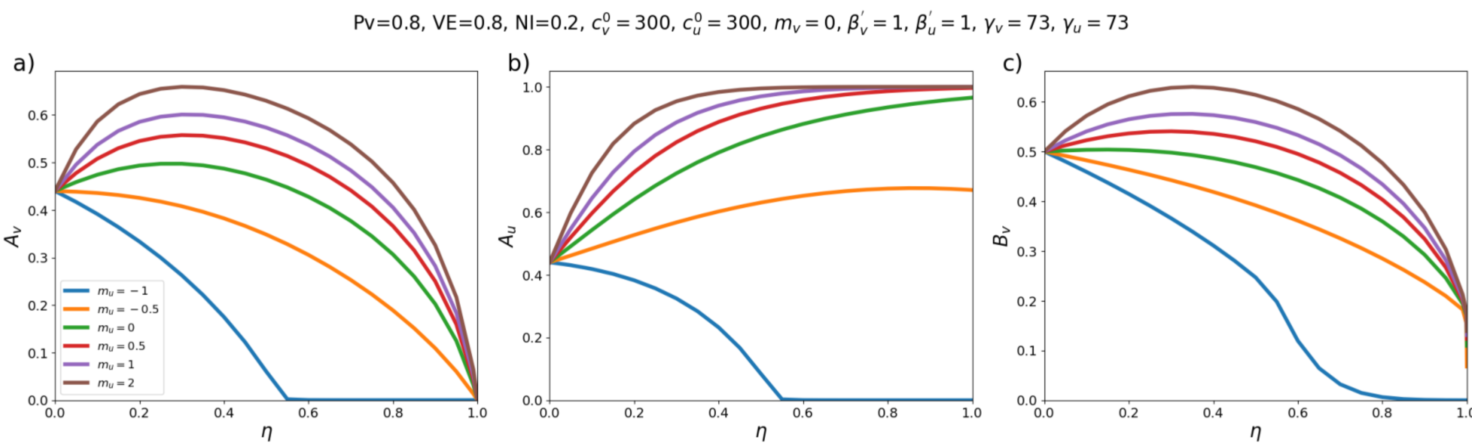


Figure 1: Three different quantities as functions of the degree of segregation, η, for different values of m_u: (a) Attack rate among the vaccinated sub-population, (b) Attack rate among the unvaccinated sub-population, (c) Share of vaccinated infections that were due to contacts with unvaccinated people. Values of fixed model parameters are indicated at the top of the figure.

Figs. 2 and 3 show results for larger $c_v^0 = c_u^0$. Compared to Fig. 1a, A_v in Figs. 2a and 3a does not increase much with η when $m_u > 0$, and A_v no longer has a maximum when $m_u = 0$. Comparing with Fig. 1a, it can also be seen that A_v increases with increasing $c_v^0 = c_u^0$ when there is no segregation (η = 0).

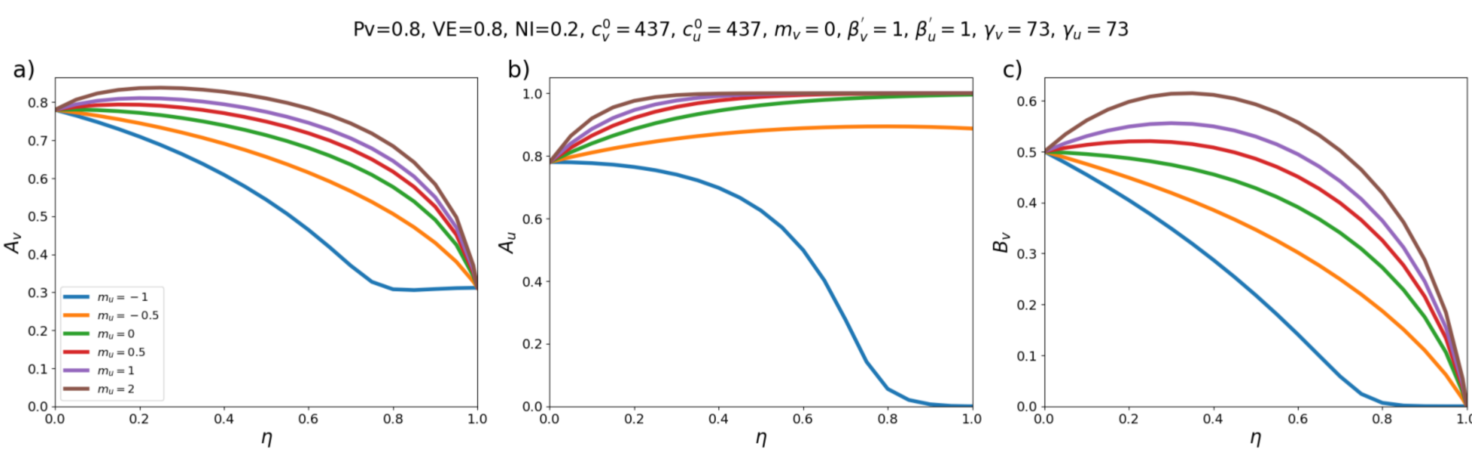


Figure 2: Same as Fig. 1, except that $c_v^0 = c_u^0 = 437$.

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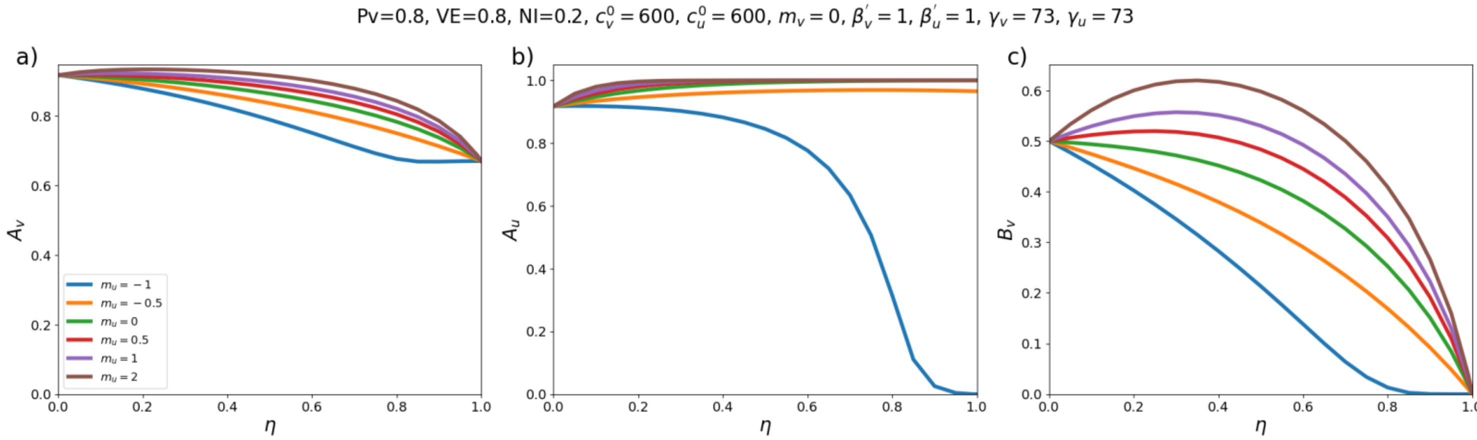


Figure 3: Same as Fig. 1, except that $c_v^0 = c_u^0 = 600$.

For smaller $c_v^0 = c_u^0$ (Figs. 4 and 5), $A_v(\eta = 0)$ is decreased, and larger η can dramatically increase A_v. Even with an isolating segregation policy ($m_u = -0.5$ in Fig. 4a), A_v is increased for moderate values of η.

When $c_v^0 = c_u^0$ are small enough ($c_v^0 = c_u^0 = 200$ in Fig. 5), there is no epidemic among the vaccinated in the absence of segregation ($A_v(\eta = 0) = 0$). However, a non-zero vaccinated-population attack rate ($A_v > 0$) occurs if η is sufficiently large, and emerges regardless of whether one isolates or compounds the unvaccinated. Therefore, for small values of $c_v^0 = c_u^0$, any type of segregation can only harm the vaccinated.

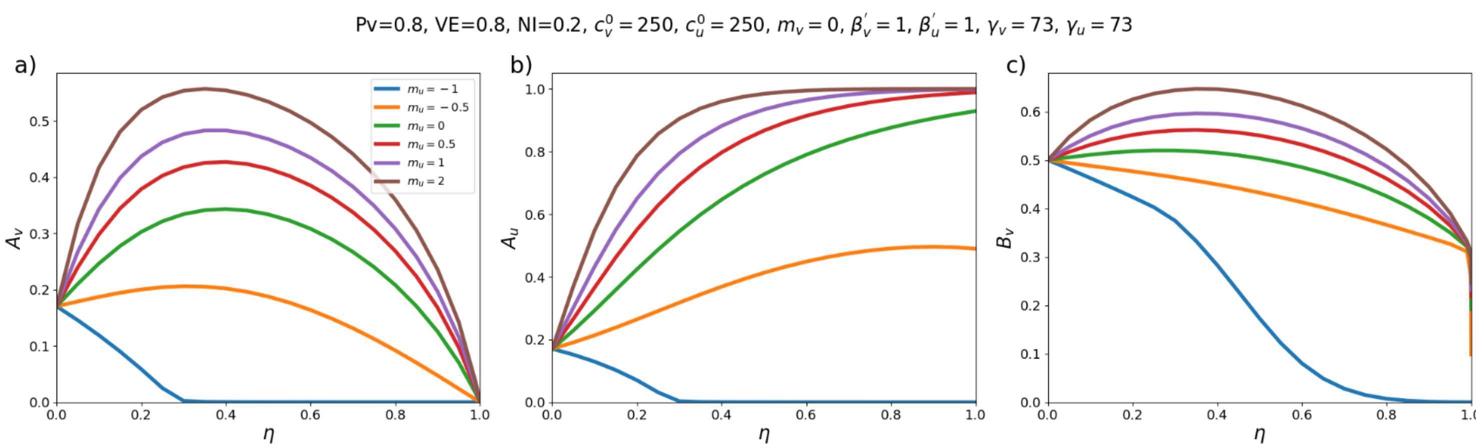


Figure 4: Same as Fig. 1, except that $c_v^0 = c_u^0 = 250$.

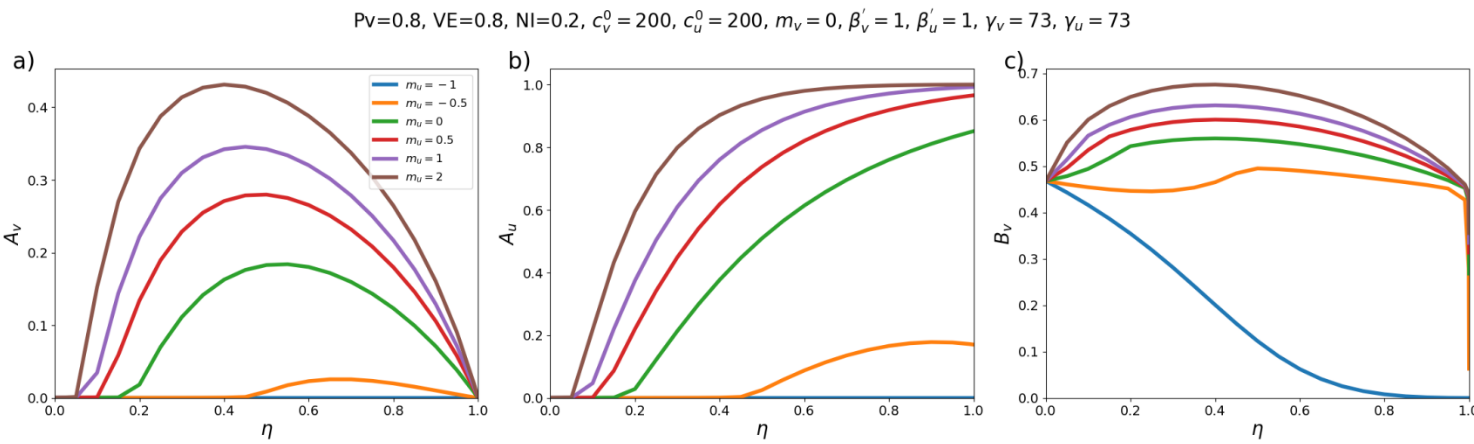


Figure 5: Same as Fig. 1, except that $c_v^0 = c_u^0 = 200$.

Appendix 2, contains supplementary figures showing that when VE is decreased (e.g. VE=0.4), A_v is not strongly influenced by η, regardless of m_u; therefore, any beneficial effect of segregation on A_v is reduced as VE decreases.

Appendix 2 also explores $c_v^0 \neq c_u^0$. For example, when $c_v^0 > c_u^0$, the unvaccinated contact frequency is reduced even when there is no segregation; increasing η can then increase A_v significantly for parameter values for which a similar increase of A_v does not occur when $c_v^0 = c_u^0$ (e.g. $c_v^0 = 437$, $m_u = 1$, $m_v = 0$, VE = 0.8, in Section A2.6).

Interpretation

Our model shows that vaccination-status-based segregation can have significantly different and counter-intuitive impacts on the outcome of an epidemic, depending on how the segregation is applied, and depending on cultural and population-density factors, for example, that co-determine c_v^0 and c_u^0.

Regarding segregation, the key feature is that the contact frequencies of people in each of the segregated sub-populations depend on the degree and type of segregation applied. Segregation that compounds the unvaccinated ($m_u > 0$ and $m_v = 0$) generally causes an increase in the vaccinated-population attack rate, A_v, for small and intermediate degrees of segregation, η. Segregation that isolates and excludes the unvaccinated ($m_u < 0$ and $m_v = 0$) decreases A_v for "more contagious viruses" (i.e. large $c_v^0 = c_u^0$); however, for "less contagious viruses" (smaller $c_v^0 = c_u^0$), both isolating and compounding types of segregation can increase A_v beyond its value in an unsegregated society. For "viruses that are not very contagious" (small $c_v^0 = c_u^0$), applying segregation can cause a sizeable epidemic among the vaccinated even though virtually no vaccinated people would be infected in an unsegregated society.

We find that B_v, the share of vaccinated infections that are due to contact with unvaccinated people, follows a similar behaviour to A_v as a function of the degree of segregation, when

segregation has no impact on the vaccinated contact frequency ($m_v = 0$). For this type of segregation, A_v and B_v either increase or decrease simultaneously with increasing η, depending on the value of m_u, and B_v is minimized for complete segregation. When $m_v = 0$, there is therefore no type or degree of segregation that reduces the vaccinated attack rate while simultaneously "enhancing the degree of risk" to vaccinated people from unvaccinated people (Fisman et al., 2022).

In contrast, when $m_v \neq 0$, such that segregation affects the contact frequencies of vaccinated people, segregation can produce an increased A_v along with a decreased B_v and vice-versa, as shown in Appendix 2 (Sections A2.3 and A2.4).

The broad range of results emerging from our simple model highlights the importance of the impact of segregation on contact frequencies, which has not been considered in other epidemic models, including network-based models in which unvaccinated people cluster together in "cliques" or households (Salathé & Bonhoeffer, 2007; De Leon & Aran, 2022; Achitouv, 2022).

Limitations

SIR models and their variations, including agent-based versions (Hinch et al., 2021; Achitouv, 2022) are based on the paradigm of transmission due to pairwise contact between a recently infected and a susceptible individual. However, this paradigm is unable to account for important features of viral respiratory disease incidence, in particular its seasonal pattern that is strongly dependent on latitude and its rapid emergence and disappearance occurring at essentially the same time at widely dispersed locations (Hope-Simpson, 1992). Seasonality of viral respiratory disease may be driven by the seasonality of absolute humidity and its effect on transmission via aerosols (Shaman & Kohn, 2009; Shaman et al., 2010). However air-borne transmission via long-lived suspended aerosol particles is not directly compatible with pairwise transmission, since it occurs in built environments where many people may transit or be present (Bulfone et al., 2021). These fundamental limitations of present viral respiratory disease models are caveats to any use of such models in health policy.

Conclusion

Using SIR modelling, we have shown that vaccination-status-based segregation can lead to significantly different and counter-intuitive epidemic outcomes depending on how segregation is applied, and depending on complex cultural and physical factors that co-determine infectious contact frequencies (i.e., the products βc), including negative health consequences for either segregated group, even disregarding the expected deleterious health impacts of the segregation policies themselves (Cohen, 2004; Cohen et al., 1991; Cohen et al., 1997). Given the lack of reliable empirical evaluations of needed infectious contact frequency values, given the now proven outcome sensitivities to the infectious contact frequencies, and given the intrinsic limitations of SIR models in this application, we cannot recommend that SIR modelling be used to motivate or justify segregation policies regarding viral respiratory diseases, in the present state of knowledge.

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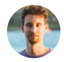
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Marine Baudin



Jérémie Mercier
Santé Liberté

COVID-Period Mass Vaccination Campaign and Public Health Disaster in the USA

From age/state-resolved all-cause mortality by time,
age-resolved vaccine delivery by time, and socio-geo-economic data

Denis G. Rancourt[1,2,*], PhD, Marine Baudin[3], PhD, Jérémie Mercier[3], PhD

[1] Correlation Research in the Public Interest (correlation-canada.org)
[2] Ontario Civil Liberties Association (ocla.ca)
[3] Mercier Production (jeremie-mercier.com)
* denis.rancourt@alumni.utoronto.ca

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2 August 2022

Abstract

All-cause mortality by time is the most reliable data for detecting and epidemiologically characterizing events causing death, and for gauging the population-level impact of any surge or collapse in deaths from any cause. Such data is not susceptible to reporting bias or to any bias in attributing causes of death. We compare USA all-cause mortality by time (month, week), by age group and by state to number of vaccinated individuals by time (week), by injection sequence, by age group and by state, using consolidated data up to week-5 of 2022 (week ending on February 5, 2022), in order to detect temporal associations, which would imply beneficial or deleterious effects from the vaccination campaign. We also quantify total excess all-cause mortality (relative to historic trends) for the entire covid period (WHO 11 March 2020 announcement of a pandemic through week-5 of 2022, corresponding to a total of 100 weeks), for the covid period prior to the bulk of vaccine delivery (first 50 weeks of the defined 100-week covid period), and for the covid period when the bulk of vaccine delivery is accomplished (last 50 weeks of the defined 100-week covid period); by age group and by state.

We find that the COVID-19 vaccination campaign did not reduce all-cause mortality during the covid period. No deaths, within the resolution of all-cause mortality, can be said to have been averted due to vaccination in the USA. The mass vaccination campaign was not justified in terms of reducing excess all-cause mortality. The large excess mortality of the covid period, far above the historic trend, was maintained throughout the entire covid period irrespective of the unprecedented vaccination campaign, and is very strongly correlated (r = +0.86) to poverty, by state; in fact, proportional to poverty. It is also correlated to several other socio-economic and health factors, by state, but not correlated to population fractions (65+, 75+, 85+ years) of elderly state residents.

The excess all-cause mortality by age group (also expressed as percentage of pre-covid-period all-cause mortality for the age group) for the whole USA for the entire covid period through week-5 of 2022 is:

all ages	1.27M	23%
0-24	13K	12%
25-44	109K	41%
45-64	274K	27%
65-74	319K	30%
75-84	316K	24%
85+	240K	14%

The corresponding fatality risk ratios are relatively uniform with age (non-exponential and non-near-exponential with age; and even skewed towards young adults), which holds essentially for all states, and for all examined periods within the covid period. This fundamental result implies that a dominant cause of excess mortality could not have been assigned COVID-19, which consistently has been measured to have a strong near-exponential infection fatality ratio with age. The implication is further corroborated by the absence of correlation between all-age-group-integrated excess mortality and age, by state. COVID-19 was not a dominant cause of excess mortality during the covid period in the USA.

All of our observations can be coherently understood if we interpret that the covid-period socio-economic, regulatory and institutional conditions induced chronic stress and social isolation among members of large vulnerable groups (individuals afflicted and co-afflicted by poverty, obesity, diabetes, high susceptibility to bacterial respiratory infection [inferred from pre-covid-period antibiotic prescription rates], old age, societal exclusion, unemployment, drug and substance abuse, and mental disability or serious mental illness), which in turn caused many of these individuals to be more and fatally immunocompromised, allowing them to succumb to bacterial pneumonia, at a time when a documented national pneumonia epidemic raged and antibiotic prescriptions were systemically reduced; in addition to possible comorbidity from COVID-19 vaccine challenge against individuals thus made immunocompromised, under broad and hastily implemented “vaccine equity” programs.

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Table of abbreviations and definitions

Abbreviation	Name	Units	Description	Notes
65+	65+	People	Resident population estimate of people aged 65 years old and over as of July 1st, 2020	
65+/pop	65+ by population	%	Proportion of the population aged 65 years old and over	
75+	75+	People	Resident population estimate of people aged 75 years old and over as of July 1st, 2020	
75+/pop	75+ by population	%	Proportion of the population aged 75 years old and over	
85+	85+	People	Resident population estimate of people aged 85 years old and over as of July 1st, 2020	
85+/pop	85+ by population	%	Proportion of the population aged 85 years old and over	
ACM	All-cause mortality	Deaths	Mortality from all causes of death (occurring in a defined period and for a defined place)	
ACM/m	ACM by month	Deaths/m	ACM occurring per month	
ACM/w	ACM by week	Deaths/w	ACM occurring per week	
At least 1 dose	At least 1 dose	People	Total count of people with at least one dose	1
Booster	Booster	People	Total count of people aged 12 years and older with a booster dose	1
CDC	Centers for Disease Control and Prevention	N/A	The Centers for Disease Control and Prevention is the national public health agency of the United States.	
COVID-19	coronavirus disease 2019	N/A	"Coronavirus disease 2019 is a contagious disease caused by severe acute respiratory syndrome coronavirus 2"	
covid period	covid period		Period starting with the WHO announcement of a pandemic on March 11, 2020, up to and including the most reliable ACM data (through December	

			2021 for the data by month; through week-5 of 2022 for the data by week)	
cvp1	COVID-peak 1	Deaths	ACM peak occurring over March, April and May 2020	
cvp2	COVID-peak 2	Deaths	ACM peak occurring over the winter 2020-2021	
Disability	Disability	%	Percent of Americans with a disability	2
Fully vaccinated	Fully vaccinated	People	Total count of people who are fully vaccinated	1
m22c	ACM for the 22-month covid period	Deaths	Integrated ACM from March 2020 to December 2021, included	
m22c-1	ACM for the 1st 22-month period prior to the covid period	Deaths	Integrated ACM from May 2018 to February 2020, included	
m22c-2	ACM for the 2nd 22-month period prior to the covid period	Deaths	Integrated ACM from July 2016 to April 2018, included	
MHI	Median Household Income	$	Estimated median household income in US dollars	
Obesity	Obesity	%	Prevalence of self-reported obesity among U.S. adults (BRFSS (Behavioral Risk Factor Surveillance System), 2020)	
pCVD	pre-covid	Deaths	corresponds to w50c-2	
pop	Population	People	Resident population estimate for the states of the USA as of July 1st of 2020	
Poverty	Poverty	%	Percent of the population living in poverty	
pVax	pre-vaccination	Deaths	corresponds to w50c-1	
pVax-pCVD	Excess mortality during the pre-vaccination period of the covid period	Deaths	pVax-pCVD = w50c-1 - w50c-2	3
pVax-pCVD/pCVD	pVax-pCVD expressed as a percentage of pre-covid mortality	%	pVax-pCVD/pCVD = (w50c-1 - w50c-2) / w50c-2 (Equation 9)	4
smp1	Summer-peak 1	Deaths	ACM peak occurring over the summer 2020	
smp2	Summer-peak 2	Deaths	ACM peak occurring over the late-summer and fall	5

			2021	
SSDI	Social Security Disability Insurance	People	Number of all disabled beneficiaries aged 18-64 of the SSDI program	
SSDI/pop	SSDI by population	%	SSDI normalized by population	
SSI	Supplemental Security Income	People	Number of recipients of the SSI program	
SSI/pop	SSI by population	%	SSI normalized by population	
USA	United States of America	N/A	USA is composed of 51 states, including the District of Columbia, Alaska and Hawaii	
VAERS	Vaccine Adverse Event Reporting System	N/A	United States program for vaccine safety, co-managed by the U.S. Centers for Disease Control and Prevention (CDC) and the Food and Drug Administration (FDA)	
Vax	vaccination	Deaths	corresponds to w50c	
Vax-pCVD	Excess mortality during the vaccination period of the covid period	Deaths	Vax-pCVD = w50c - w50c-2	6
Vax-pCVD/pCVD	Vax-pCVD expressed as a percentage of pre-covid mortality	%	Vax-pCVD/pCVD = (w50c - w50c-2) / w50c-2 (Equation 10)	7
Vax-pVax	Difference in mortality between the vaccination period and the pre-vaccination period of the covid period	Deaths	Vax-pVax = w50c - w50c-1 (Equation 11)	
Vax-pVax/pCVD	Vax-pVax expressed as a percentage of pre-covid mortality	%	Vax-pVax/pCVD = (w50c - w50c-1) / w50c-2 (Equation 12)	
w100c	ACM for the 100-week covid period	Deaths	Integrated ACM from week-11 of 2020 (week of March 9, 2020) to week-5 of 2022 (week of January 31, 2022), included	
w100c-1	ACM for the 1st 100-week period prior to the covid	Deaths	Integrated ACM from week-15 of 2018 (week of April 9, 2018) to week-10 of 2020 (week of March 2,	

	period		2020), included	
w100c-2	ACM for the 2nd 100-week period prior to the covid period	Deaths	Integrated ACM from week-19 of 2016 (week of May 9, 2016) to week-14 of 2018 (week of April 2, 2018), included	
w50c	ACM for the 50-week vaccination period of the covid period	Deaths	Integrated ACM from week-8 of 2021 (week of February 22, 2021) to week-5 of 2022 (week of January 31, 2022), included	8
w50c-1	ACM for the 50-week pre-vaccination period of the covid period	Deaths	Integrated ACM from week-11 of 2020 (week of March 9, 2020) to week-7 of 2021 (week of February 15, 2021), included	9
w50c-2	ACM for the 1st 50-week period prior to the covid period	Deaths	Integrated ACM from week-13 of 2019 (week of March 25, 2019) to week-10 of 2020 (week of March 2, 2020), included	10
WHO	World Health Organization	N/A	The World Health Organization is a specialized agency of the United Nations responsible for international public health.	
xDc(22)1	Excess mortality during the 22-month covid period, relative to m22c-1	Deaths	xDc(22)1 = m22c - m22c-1 (Equation 1)	
xDc(22)1%	xDc(22)1 expressed as a percentage of pre-covid mortality	%	xDc(22)1% = xDc(22)1 / m22c-1 (Equation 3)	
xDc(22)2	Excess mortality during the 22-month covid period, relative to m22c-2	Deaths	xDc(22)2 = m22c - m22c-2 (Equation 2)	
xDc(22)2%	xDc(22)2 expressed as a percentage of pre-covid mortality	%	xDc(22)2% = xDc(22)2 / m22c-2 (Equation 4)	
xDc(100)1	Excess mortality during the 100-week covid period, relative to w100c-1	Deaths	xDc(100)1 = w100c - w100c-1 (Equation 5)	11
xDc(100)1%	xDc(100)1 expressed as a percentage of pre-covid	%	xDc(100)1% = xDc(100)1 / w100c-1 (Equation 7)	12

	mortality			
xDc(100)1/pop	xDc(100)1 by population		xDc(100)1 normalized by population	13
xDc(100)2	Excess mortality during the 100-week covid period, relative to w100c-2	Deaths	xDc(100)2 = w100c - w100c-2 (Equation 6)	
xDc(100)2%	xDc(100)2 expressed as a percentage of pre-covid mortality	%	xDc(100)2% = xDc(100)2 / w100c-2 (Equation 8)	

1 In Figures 10 and 11, it is presented as the cumulative number of people by week
2 Disability is defined as a long-lasting sensory, physical, mental, or emotional condition or conditions that make it difficult for a person to do functional or participatory activities such as seeing, hearing, walking, climbing stairs, learning, remembering, concentrating, dressing, bathing, going outside the home, or working at a job.
3 Also called "pre-vaccination-period excess mortality" in the text
4 Also called "covid-period pre-vaccination-period relative excess mortality" in the text
5 Also called "late-summer-2021 peak" in the text
6 Also called "vaccination-period excess mortality" in the text
7 Also called "covid-period vaccination-period relative excess mortality" in the text
8 Also called "integrated mortality in the vaccination period of the covid period" in the text
9 Also called "integrated mortality in the pre-vaccination period of the covid period" in the text
10 Also called "pre-covid-period integrated mortality" in the text
11 Also called "100-week covid-period excess mortality" in the text
12 Also called "covid-period fatality risk ratio" in the text
13 Also called "100-week covid-period fatality ratio" in the text
N/A stands for not applicable

1. Introduction

Following Rancourt's 2 June 2020 article critically assessing circumstances of the declared pandemic using all-cause mortality (ACM) (Rancourt, 2020), more and more researchers are recognizing that it is essential to examine ACM by time, and excess deaths from all causes compared with projections from historic trends, to help make sense of the events surrounding COVID-19 (Kontis *et al.*, 2020; Rancourt, Baudin and Mercier, 2020; Villani *et al.*, 2020; Rancourt, Baudin and Mercier, 2021a, 2021b; Achilleos *et al.*, 2021; Chan, Cheng and Martin, 2021; Faust *et al.*, 2021; Islam, Jdanov, *et al.*, 2021; Islam, Shkolnikov, *et al.*, 2021; Jacobson and Jokela, 2021; Joffe, 2021; Karlinsky and Kobak, 2021; Kobak, 2021; Kontopantelis *et al.*, 2021; Locatelli and Rousson, 2021; Sanmarchi *et al.*, 2021; Woolf *et al.*, 2021; Woolf, Masters and Aron, 2021; Kontopantelis *et al.*, 2022; Ackley *et al.*, 2022; Johnson and Rancourt, 2022; Lee *et al.*, 2022; Wang *et al.*, 2022).

Rancourt (2020) argued that ACM by time and by jurisdiction data for many countries and states of the USA in the months that followed the WHO 11 March 2020 declaration of a pandemic:

(1) was inconsistent with the dominant view of the characteristic features of a pandemic (high contagiousness and spread by person-to-person "contact"), and
(2) gave clear evidence of synchronous local "hot spot" (jurisdictional) response-induced mortality.

Likewise, in our further prior analyses of ACM by time (by day, week, month, year) for many countries (and by province, state, region or county), we found that both the initial and long-term ACM data in the covid period is inconsistent with a viral respiratory disease pandemic, where the time-integrated mortality per capita is highly heterogeneous between jurisdictions, with no anomalies in the first many months in most places, and hot spots or hot regions having death rate increases that are synchronous with aggressive local or regional responses, both medical and

governmental, which accompanied the 11 March 2020 WHO declaration of a pandemic (Rancourt, Baudin and Mercier, 2020, 2021a, 2021b; Johnson and Rancourt, 2022).

The initial surges in ACM are highly localized geographically (by jurisdiction) and are precisely synchronous (all starting immediately after the 11 March 2020 WHO declaration of a pandemic, across continents), which is contrary to model pandemic behaviour; but is consistent with the surges being caused by the known government and institutional responses (Rancourt, 2020; Rancourt, Baudin and Mercier, 2020, 2021a, 2021b; Johnson and Rancourt, 2022).

The ACM by time data for the USA in the covid period has extraordinary features, including large peaks occurring in the summer seasons, and dramatically different state to state behaviours. State-to-state heterogeneity in integrated covid-period health-status-adjusted mortality is well illustrated by Johnson and Rancourt (Johnson and Rancourt, 2022; their Figure 7). Above-decadal-trend mortality in the covid period is massive. Nothing like this occurs in neighbouring Canada (Rancourt, Baudin and Mercier, 2021a). Nothing like this occurs in Western European countries. Similar anomalies occur in some Eastern European countries and in Russia. The large differences in covid-period mortality in the USA compared to other Western countries are probably related to the known relatively poor health-status of the USA population, suggesting large groups of particularly vulnerable residents (Roser, 2020).

We found that in the USA the state-wise integrated excess ACM of all main age groups in the summer seasons (2020 and 2021) especially was largest (on a per capita basis) in the southern states, and was correlated to state-specific obesity and poverty rates, strongly correlated to the product of obesity and poverty rates, and correlated to mean climatic temperature of the state, and to state-wise pre-covid-period antibiotic prescription rate per capita (Rancourt, Baudin and Mercier, 2021b). We postulated that vulnerable groups became more immune-deficient due to increased experienced physio-psychological stress and social isolation, and mostly succumbed to bacterial pneumonia, which is the dominant comorbidity (40-60%) reported in the CDC covid

mortality data, at a time when antibiotic prescription rates show an unprecedented decrease (Rancourt, Baudin and Mercier, 2021b).

In the present article, we extend our epidemiological analysis using consolidated ACM data (by month, by week, by state, and by age group) up to week-5 of 2022 (week ending on February 5, 2022), which gives us 100 weeks since the WHO's 11 March 2020 declaration of a pandemic.

Our goal is three-fold:

(1) Accurately quantify excess mortality (ACM) during the covid period in the USA
(2) Look for socio-economic factors that correlate to time-integrated excess ACM per capita, by state
(3) Examine whether any impact of the COVID-19 vaccination campaign, which was implemented in 2021, can be detected and quantified

Presently (as of July 14, 2022), a total of 221,924,152 people are fully vaccinated against COVID-19 (Johns Hopkins, 2022) in a population of 332,878,208 (US Census Bureau, 2022a), following an unprecedented vaccination campaign, which was largely accomplished in the last 50 weeks of the covid period up to week-5 of 2022.

- Has this massive campaign had any measurable impact, positive or negative, on the all-cause mortality in the USA, for any discerned age group?
- Can such an impact be detected in delayed or immediate synchronicity with the dose delivery rates for the different age groups?
- Are there important differences in ACM by time and by age group for the periods (within the covid period) prior to and following vaccine dose delivery, and how should such differences be interpreted if they occur?

2. Data

Table 1 describes the data used in this work and the sources of the data.

Data	Country	Period	Time unit	Filters	Source
ACM	USA	1999-2021*	Month	State, sex, age group[1]	CDC, 2022a
ACM	USA	2015-2022**	Week	State, age group[2]	CDC, 2022b
Vaccines	USA	2020-2022⁺	Day	Age group[3]	CDC, 2022c
Vaccines	USA	2020-2022⁺⁺	Day	State, age group[4]	CDC, 2022d
Obesity	USA	2020	Year	State	CDC, 2021
Population	USA	2010-2020§	Year	State, sex, age group[5]	US Census Bureau, 2021
Poverty	USA	2020	Year	State	US Census Bureau, 2022b
MHI	USA	2020	Year	State	US Census Bureau, 2022b
SSI	USA	2020	Year	State	SSA, 2022a
SSDI	USA	2020	Year	State	SSA, 2022b
Disability	USA	-	-	State	Disabled World, 2020

Table 1. Data retrieved. In this work, USA is composed of 51 states, including the District of Columbia, Alaska and Hawaii, unless otherwise stated in the text.
* These data are a combination of the data found in CDC 2022a: data for the years 1999 to 2020 were downloaded under the “Current Final Multiple Cause of Death Data” section of the reference (on November 17, 2021 for the years 1999 to 2019 and on May 18, 2022 for the year 2020), and data for the year 2021 was downloaded under the “Provisional Multiple Cause of Death Data” section of the reference on May 18, 2022. The complete series is thus from January 1999 to December 2021.
** At the date of access, data were available from week-1 of 2015 (week ending on January 10, 2015) to week-19 of 2022 (week ending on May 14, 2022). Usable data are until week-5 of 2022

(week ending on February 5, 2022) due to unconsolidated data in later weeks, which gives a large artifact (anomalous drop in mortality).
[+] At the date of access, data were available from Sunday December 13th 2020 to Wednesday May 4th 2022.
[++] At the date of access, data were available from Sunday December 13th 2020 to Sunday April 24th 2022.
[§] In this work, we use the population data of the year 2020.
[1] 11 age groups: <1, 1-4, 5-14, 15-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85+
[2] 6 age groups: 0-24, 25-44, 45-64, 65-74, 75-84, 85+
[3] 9 age groups: <5, 5-11, 12-17, 18-24, 25-39, 40-49, 50-64, 65-74, 75+
[4] 4 age groups: 5+, 12+, 18+, 65+
[5] 86 age groups: by 1 year age group, from 0 to 85+

The vaccines data are daily cumulative data; when shown together with all-cause mortality by week data, the last day of the week is used (the Saturday) as a data point, so that both ACM and vaccination data correspond to the same time point (end of week for both).

The vaccines data presented in this work correspond to three data type (CDC, 2022c):

- *At least 1 dose*, corresponds to the "total count of people with at least one dose".
- *Fully vaccinated*, corresponds to the "total count of people who are fully vaccinated".
- *Booster*, corresponds to the "total count of people aged 12 years and older with a booster dose".

According to the CDC, a person is considered fully vaccinated when they "have second dose of a two-dose vaccine or one dose of a single-dose vaccine".

A booster dose is an additional dose given to a fully vaccinated person.

For all the scatter plots presented in this article, the following color-code is applied for the 51 states of the USA:

● California ● Texas ● Florida ● New York ● Pennsylvania ● Illinois ● Ohio ● Georgia ● North Carolina ● Michigan
● Alabama ● Alaska ● Arizona ● Arkansas ● Colorado ● Connecticut ● Delaware ● District of Columbia ● Hawaii
● Idaho ● Indiana ● Iowa ● Kansas ● Kentucky ● Louisiana ● Maine ● Maryland ● Massachusetts ● Minnesota
● Mississippi ● Missouri ● Montana ● Nebraska ● Nevada ● New Hampshire ● New Jersey ● New Mexico
● North Dakota ● Oklahoma ● Oregon ● Rhode Island ● South Carolina ● South Dakota ● Tennessee ● Utah
● Vermont ● Virginia ● Washington ● West Virginia ● Wisconsin ● Wyoming

3. Results

3.1. USA all-cause mortality by month, 1999-2021

3.1.1. Historic trend, normal pre-covid period seasonal pattern

Figure 1 shows the all-cause mortality by month (ACM/m) for the USA from January 1999 to December 2021.

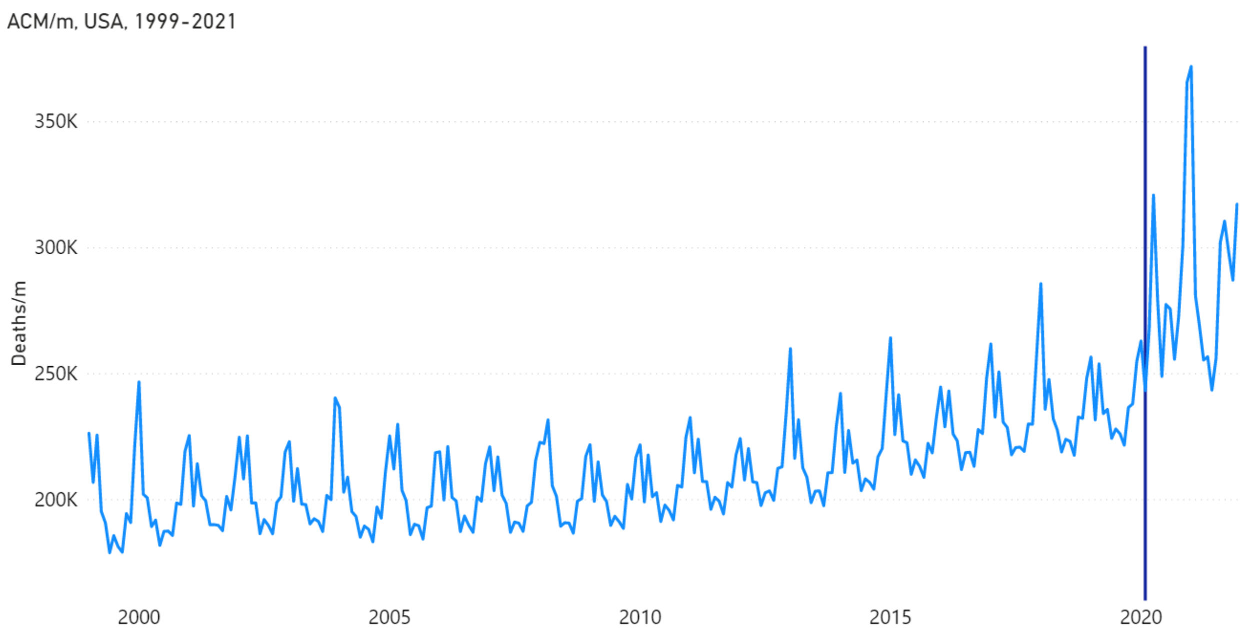


Figure 1. All-cause mortality by month in the USA from 1999 to 2021. Data are displayed from January 1999 to December 2021. The vertical dark-blue line indicates the month of February 2020, intended to point the beginning of the covid period. Data were retrieved from CDC (CDC, 2022a), as described in Table 1.

The usual seasonal variations are evident, exhibiting a regular pattern of mortality maximums in winter and mortality minimums in summer. The summer troughs follow a straight-line trend on a decadal or shorter timescale. On Figure 1 we discriminate two such periods: 2000-2008 and 2009-2019.

ACM/m has artifacts caused by the months having different numbers of days, unlike weeks (which always have 7 days). The most noticeable such artifact is the dip for the month of February, which usually has only 28 days. This allows the viewer to spot February in each winter season.

The regular seasonal pattern of mortality by month in the USA since 1999 is broken after February 2020 (Figure 1, vertical dark-blue line) when large anomalies occur. The anomalies occur in what we define as the covid period, starting after the 11 March 2020 WHO declaration of a pandemic.

We showed and discussed these anomalies in detail recently, for ACM by week (ACM/w) for the USA from week-1 (beginning of January) of 2013 to week-37 (mid-September) of 2021 (Rancourt, Baudin and Mercier, 2021b).

3.1.2. Anomalies in the covid period

In the covid period, after February 2020, we note the same peaks or features that we have previously described and interpreted (Rancourt, Baudin and Mercier, 2021b), using the nomenclature from our previous article:

- cvp1 (March-May 2020)
- smp1 (summer 2020)
- cvp2 (winter 2020-2021)
- smp2 (late-summer 2021)

Figure 2 shows those features with their labels.



Figure 2. All-cause mortality by month in the USA from 2016 to 2021. Data are displayed from July 2016 to December 2021. The cvp1, smp1, cvp2 and smp2 features discussed in the text are indicated. The vertical dark-blue line represents the month of February 2020, intended to point the beginning of the covid period. Data were retrieved from CDC (CDC, 2022a), as described in Table 1.

The anomalies in the covid period are as follows:

- A mortality peak late in the 2020 winter season, cvp1 (from February to June 2020, Figure 2).
- Peaks of mortality in the summers 2020 and 2021, smp1 and smp2, respectively, when mortality values are usually at their lowest. On Figure 2 specifically:
    - smp1 from June to September 2020
    - smp2 from July to November 2021 (connecting with the winter 2021-2022)
- A large mortality peak in the winter 2020-2021, cvp2 (from September 2020 to April 2021, Figure 2), which surpasses in magnitude any single winter mortality peaks since at least 1999 (Figure 1).

In the next section, we use the monthly ACM data to quantify the total excess mortality that occurred in the covid period, which contains these anomalies.

3.1.3. Quantifying excess mortality of the covid period, by age group and sex

We use the ACM/m data of Figure 1 to quantify the excess deaths of the covid period "to date", compared to the historic trend, as follows.

For a given age group and sex, we add all the monthly deaths together, for the months of March-2020 (start of the pandemic period; announced by the WHO on 11 March 2020) through to the latest useable month (December 2021). This is a total for 22 months (the covid period "to date"). We call this total "m22c". Then we perform a similar total for the 1st-prior 22-month period, immediately preceding the covid period, for the 22 months up to and including February 2020. We call this total "m22c-1". And we do the same for the 2nd-prior 22-month period, and we call this total "m22c-2". We continue moving back in time, to the end of the useable data in 22-month periods: m22c-3, etc.

Figure 3 shows the graph of "m22c-x" versus time, together with the ACM/m for the USA where each 22-month period has been emphasized with a different color.

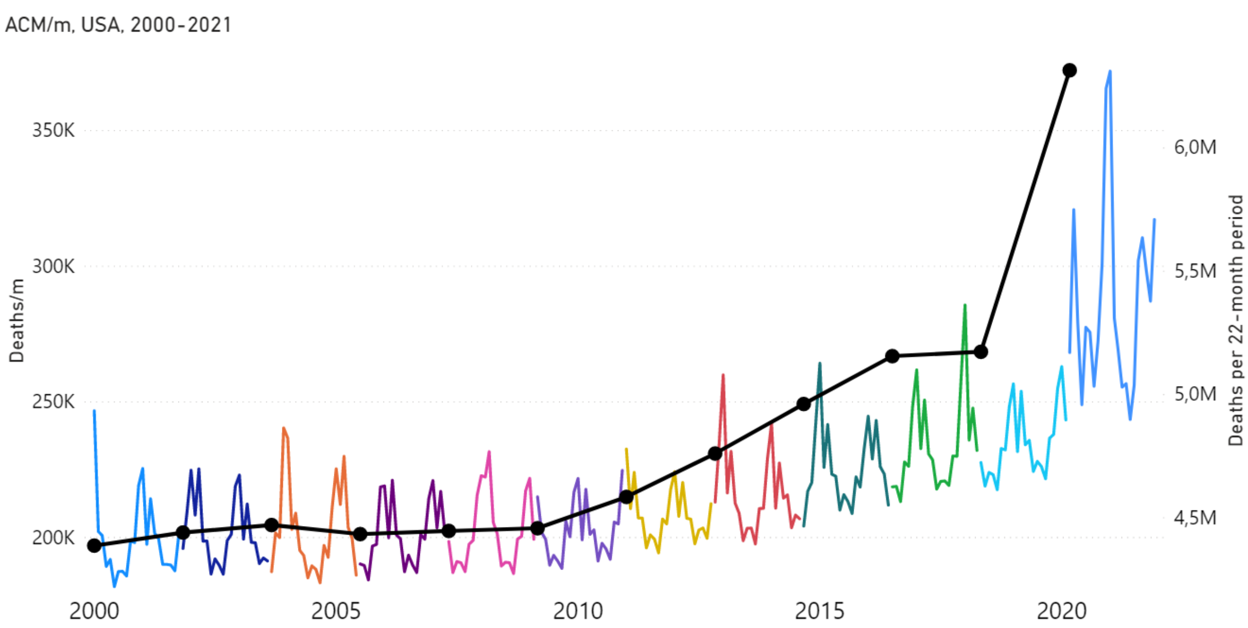


Figure 3. All-cause mortality by month (colors) and by 22-month period (black) in the USA from 2000 to 2021. Data are displayed from January 2000 to December 2021. The

different colors indicate the successive 22-month periods. The last light-blue color corresponds to the covid period. All the other previous colors are in the pre-covid period. The black line shows the integration of these successive 22-months periods. Data were retrieved from CDC (CDC, 2022a), as described in Table 1.

Figure 3, based on more than two decades of data, dramatically illustrates the sudden change in regime of ACM by time, both in magnitude of time-integrated ACM and in seasonal behaviour of ACM by time, occurring as soon as the WHO on 11 March 2020 announced a pandemic. In addition, the covid-period regime of ACM by time is characterized by large (and unprecedented in the historic record) heterogeneity by state of ACM, which is not shown in such a figure for the whole USA, but which can be appreciated in the ACM/w by state graphs of Appendix A.

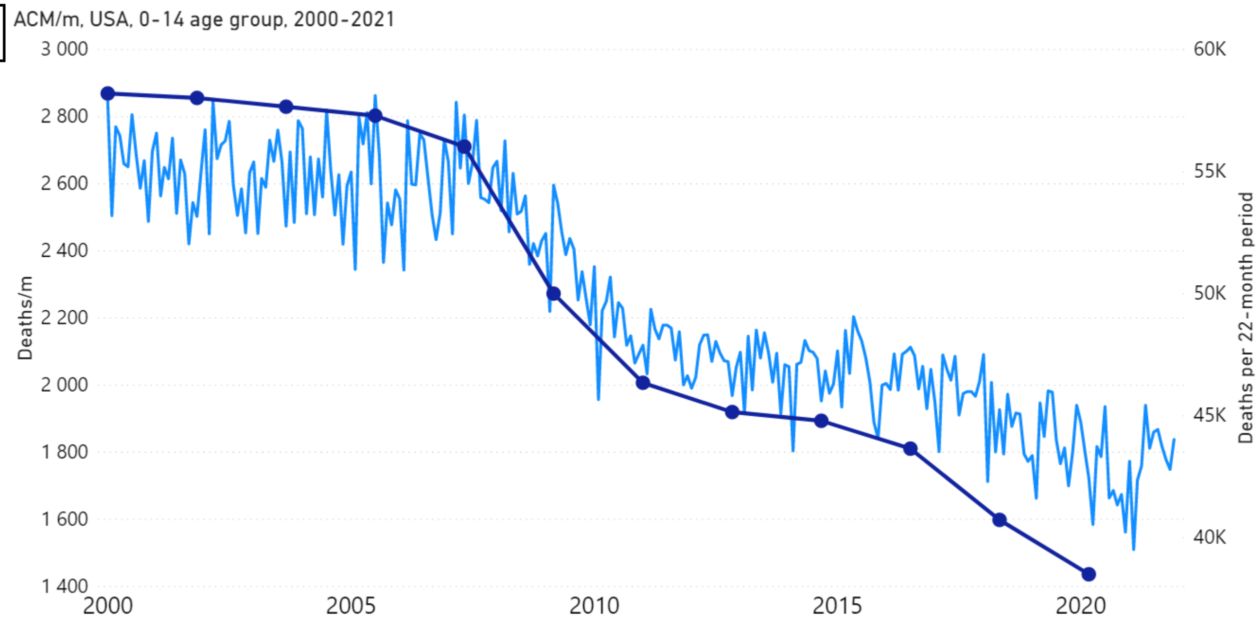
In Figure 3, each dot of the 22-month period deaths corresponds to the integration of deaths by month from the month of the dot to the previous month of the next dot, included. So the integrated deaths are shown at the beginning of each integration period (emphasized with colors).

With the integrated mortality by 22-month periods, we can spot a plateau of deaths from 2000 to 2010, an increase from 2010 to 2019, and the break between the pre-covid period and the covid period (2020).

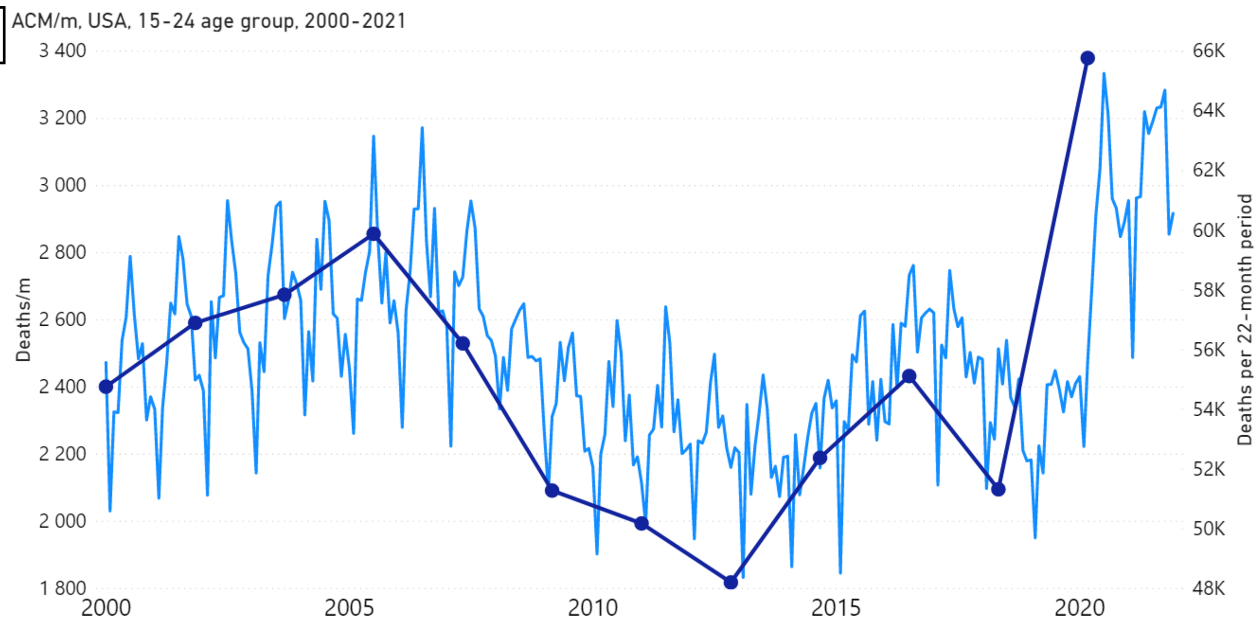
Figure 4 shows the integration of the 22-month periods with ACM/m for each of the 10-year age groups.

Figure 4. All-cause mortality by month (light-blue) and by 22-month period (dark-blue) in the USA from 2000 to 2021, for each of the age groups. Data are displayed from January 2000 to December 2021. Panels below: (A) for the 0-14 years age group; (B) for the 15-24 years age group; (C) for the 25-34 years age group; (D) for the 35-44 years age group; (E) for the 45-54 years age group; (F) for the 55-64 years age group; (G) for the 65-74 years age group; (H) for the 75-84 years age group; (I) for the 85+ years age group. Data were retrieved from CDC (CDC, 2022a), as described in Table 1.

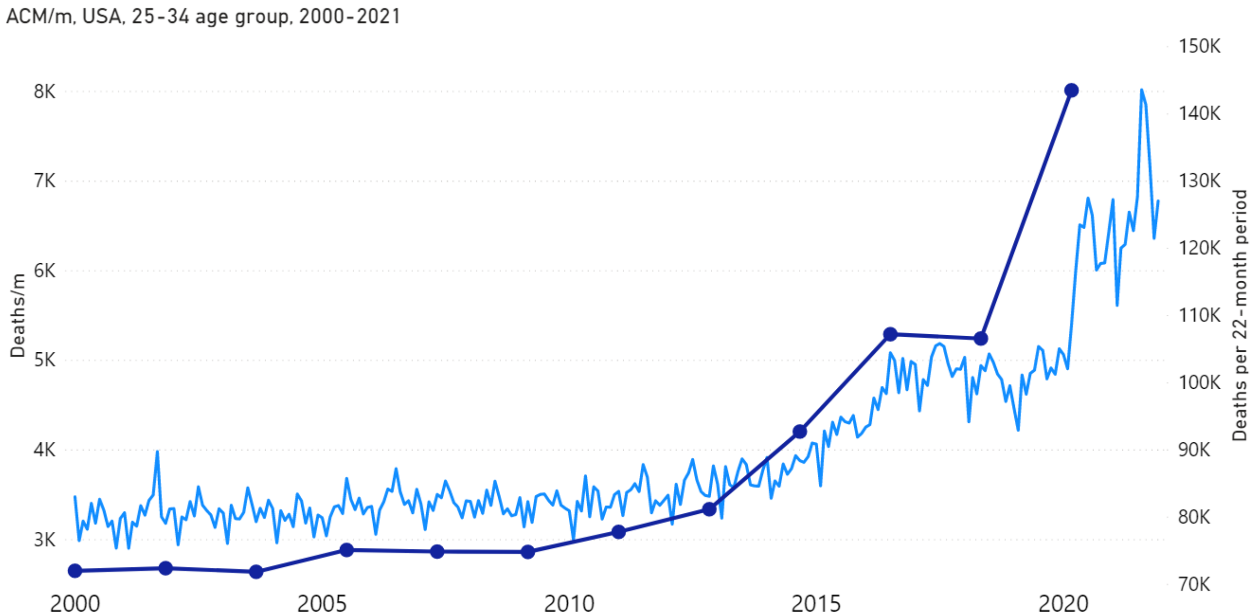
A


ACM/m, USA, 0-14 age group, 2000-2021
Deaths/m
Deaths per 22-month period
3 000
2 800
2 600
2 400
2 200
2 000
1 800
1 600
1 400
60K
55K
50K
45K
40K
2000
2005
2010
2015
2020

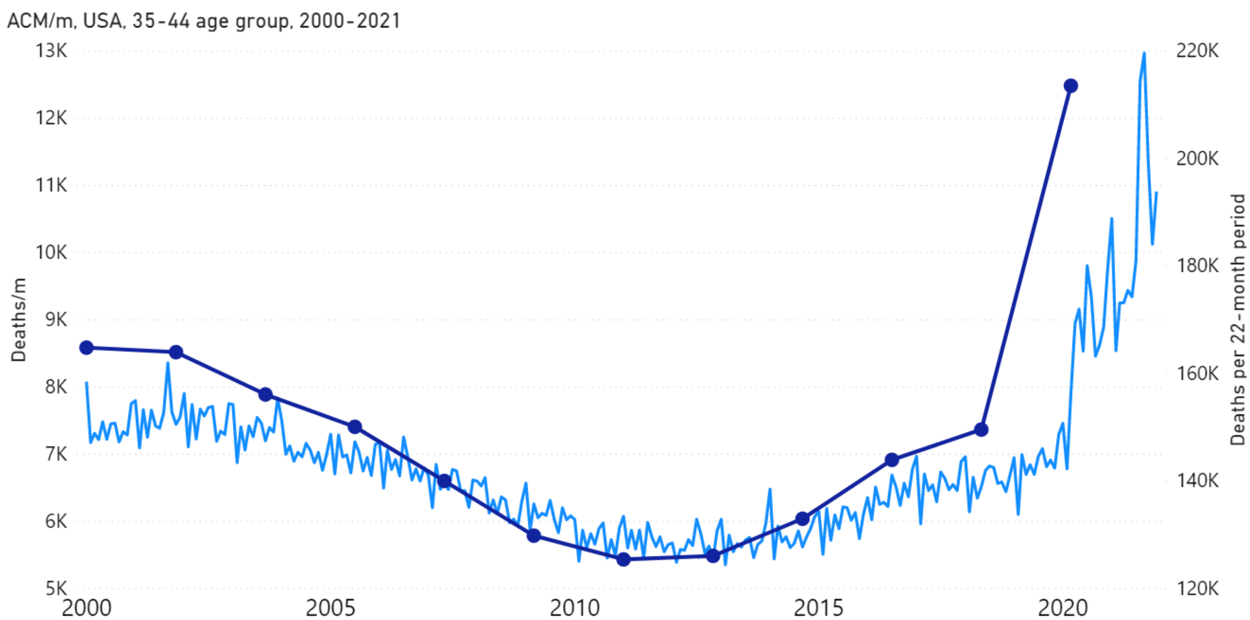
B


ACM/m, USA, 15-24 age group, 2000-2021
Deaths/m
Deaths per 22-month period
3 400
3 200
3 000
2 800
2 600
2 400
2 200
2 000
1 800
66K
64K
62K
60K
58K
56K
54K
52K
50K
48K
2000
2005
2010
2015
2020

C

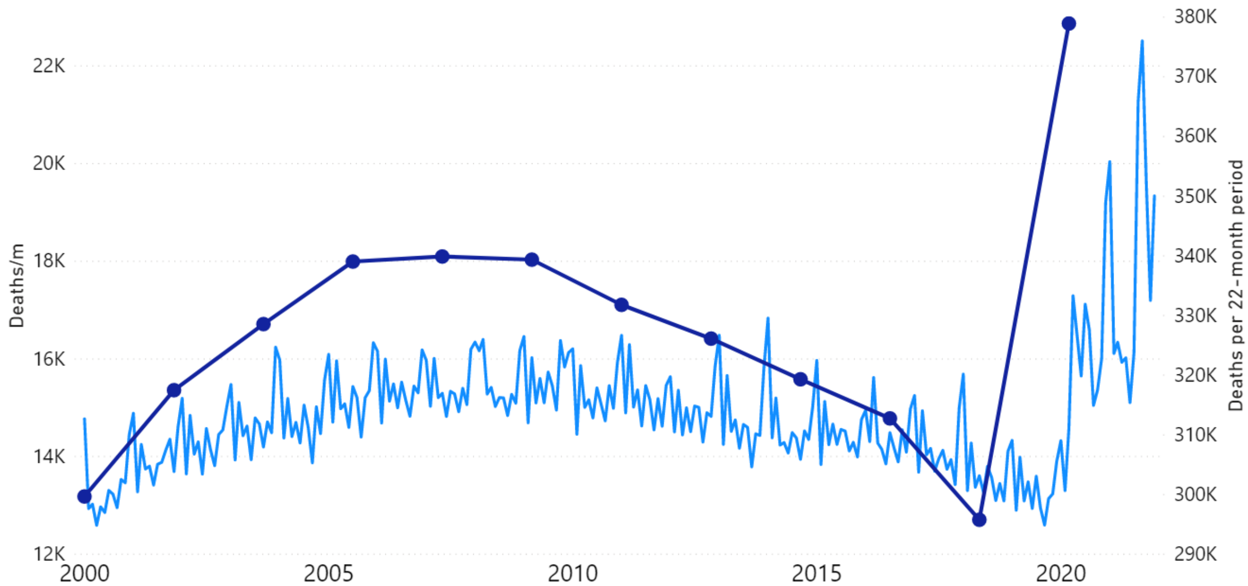

ACM/m, USA, 25-34 age group, 2000-2021
Deaths/m
8K
7K
6K
5K
4K
3K
150K
140K
130K
120K
110K
100K
90K
80K
70K
Deaths per 22-month period
2000
2005
2010
2015
2020

D


ACM/m, USA, 35-44 age group, 2000-2021
Deaths/m
13K
12K
11K
10K
9K
8K
7K
6K
5K
220K
200K
180K
160K
140K
120K
Deaths per 22-month period
2000
2005
2010
2015
2020

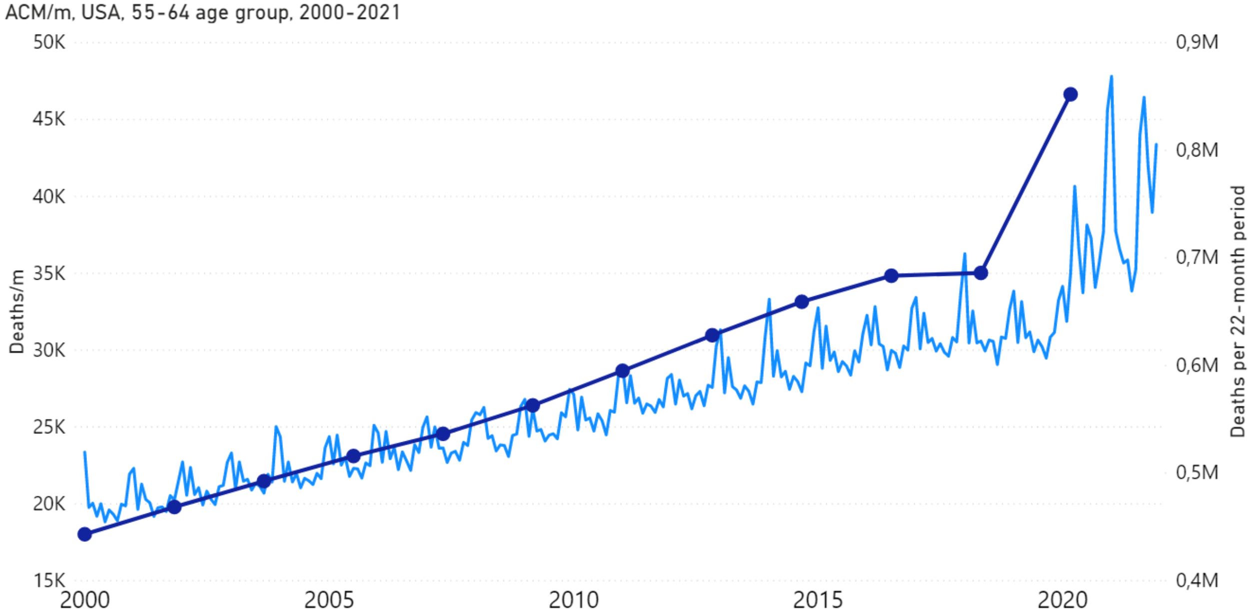
E

ACM/m, USA, 45-54 age group, 2000-2021

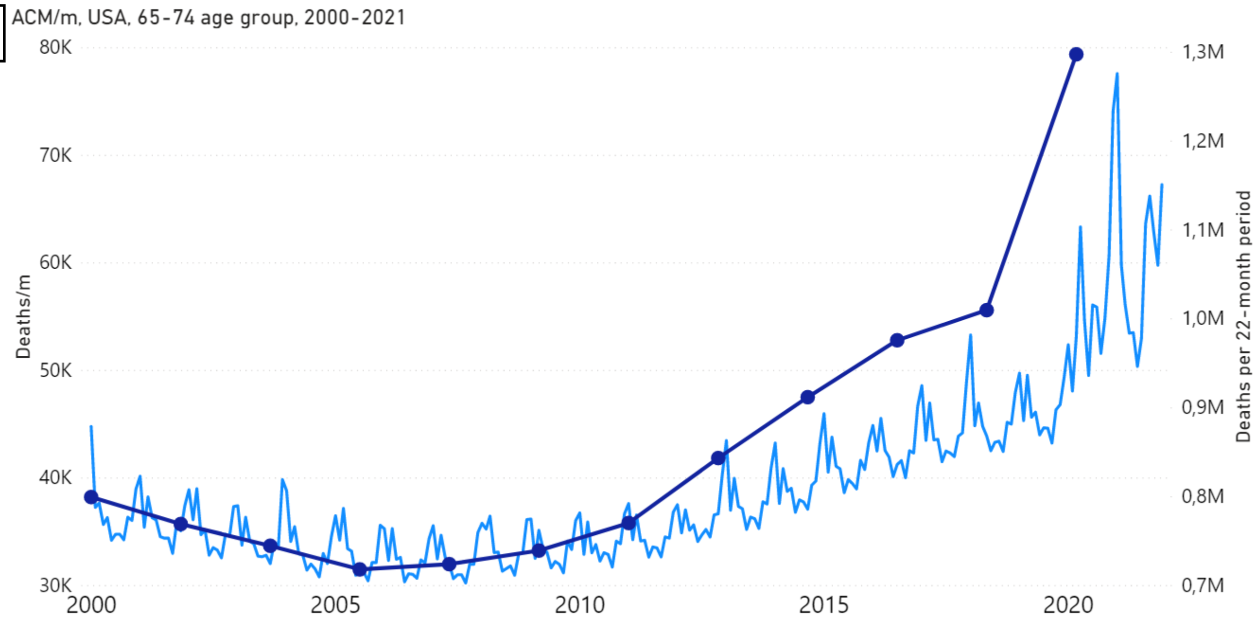


F

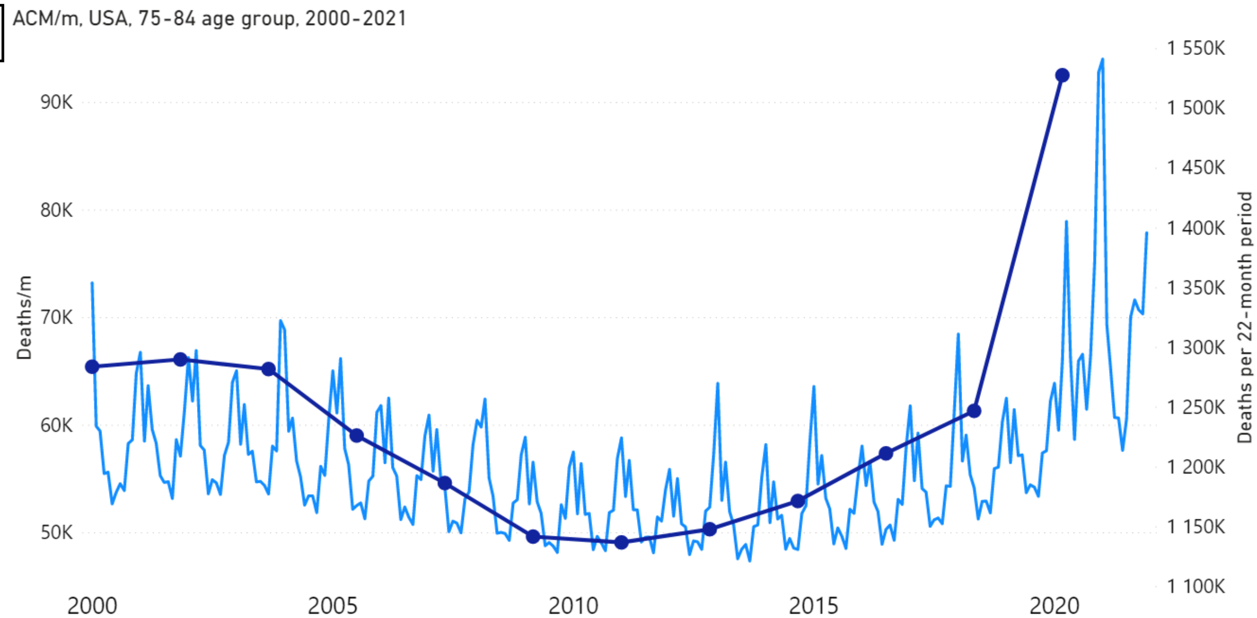
ACM/m, USA, 55-64 age group, 2000-2021



G


ACM/m, USA, 65-74 age group, 2000-2021
80K
70K
60K
50K
40K
30K
Deaths/m
1,3M
1,2M
1,1M
1,0M
0,9M
0,8M
0,7M
Deaths per 22-month period
2000
2005
2010
2015
2020

H


ACM/m, USA, 75-84 age group, 2000-2021
90K
80K
70K
60K
50K
Deaths/m
1 550K
1 500K
1 450K
1 400K
1 350K
1 300K
1 250K
1 200K
1 150K
1 100K
Deaths per 22-month period
2000
2005
2010
2015
2020

I

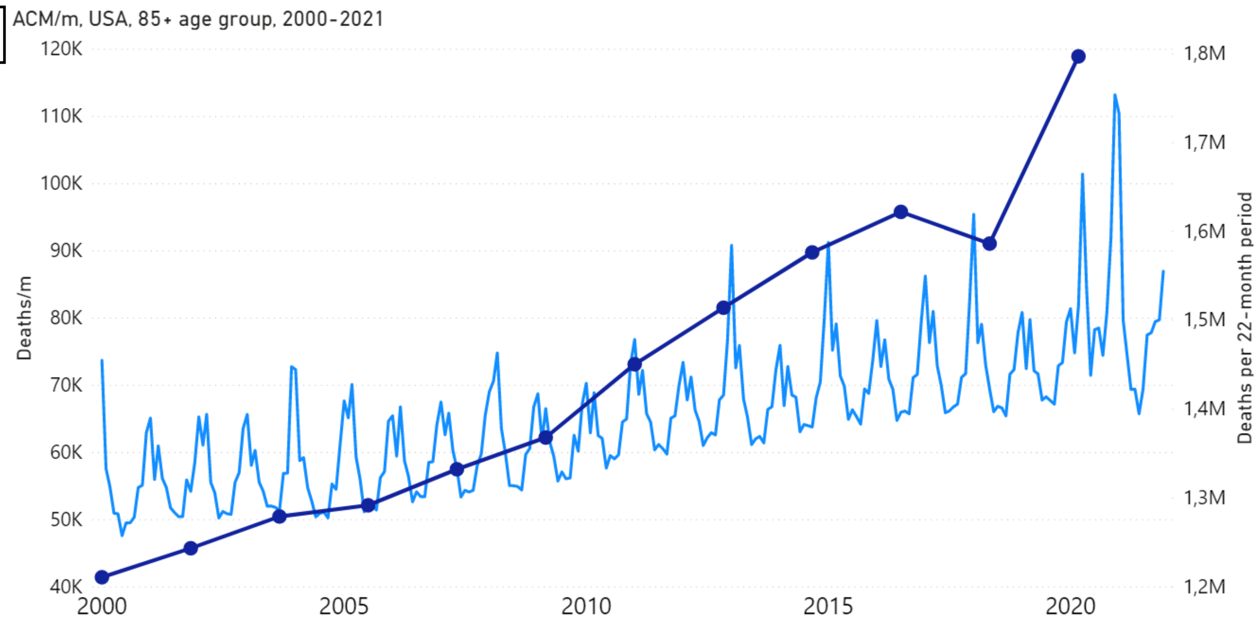


Figure 4 is hard raw data that allows one to robustly evaluate a covid-period fatality risk (covid-period excess mortality compared to the historic trend of pre-covid-period mortality) for each age group. Here, with an eye to more than two decades of data for the whole USA.

Except for the younger age group (the 0-14 year-olds, Figure 4A), we can see the break in mortality from the covid period for all the age groups: mortality by month reaches a new higher plateau and mortality by 22-month period has an increase beyond the one expected from the historic trend (Figure 4B, C, D, E, F, G, H, I). This is especially true for the 25-34 and 35-44 year-olds (Figure 4C and D), which experience close to a 50% increase in the covid period compared to the period of same duration immediately before.

Next, for a given age group and sex, we calculate the excess deaths of the covid period “to date” using two different assumptions, as follows.

In the first assumption, we take the “excess deaths of the covid period” to mean the (all-cause) deaths above the deaths that would have occurred if the same circumstances

would have prevailed during the covid period as prevailed in the 1st-prior 22-month period, immediately preceding the covid period. Under this assumption, the excess deaths of the covid period, due to everything different or extraordinary that occurred or was imposed during the covid period, for a given age group and sex, is simply:

$$xDc(22)1 = m22c - m22c\text{-}1 \qquad (1)$$

In the second assumption, we take the “excess deaths of the covid period” to mean the (all-cause) deaths above the deaths that would have occurred if the same circumstances would have prevailed during the covid period as prevailed in the 2nd-prior 22-month period, the period preceding the 1st-prior 22-month period before the covid period. Under this assumption, the excess deaths of the covid period, due to everything different or extraordinary that occurred or was imposed during the covid period, for a given age group and sex, is:

$$xDc(22)2 = m22c - m22c\text{-}2 \qquad (2)$$

These formulas (Equations 1 and 2) are justified because m22c-1 and m22c-2 are different and fair estimates of what mortality would have been in the 22-month covid period if the events associated with the declared pandemic had not occurred. In other words, m22c-1 and m22c-2 are fair historical projected values of what the covid-period mortality “would have been”. Judging from Figure 3 and Figure 4, there would be little benefit from applying a more mathematically sophisticated extrapolation method, while using both reference values allows one to estimate the uncertainty in our determinations of excess mortality for the covid period.

The relative magnitudes of the covid-period extra deaths above the historic trend are:

$$xDc(22)1\% = xDc(22)1 \ / \ m22c\text{-}1, \text{ expressed as a percentage,} \qquad (3)$$

and

$$xDc(22)2\% = xDc(22)2 \ / \ m22c\text{-}2, \text{ expressed as a percentage,} \qquad (4)$$

Table 2 contains the calculated covid-period excess mortality, for each age group and sex for the USA, and for all ages and both sexes for the entire USA (“Total”), using each assumption described above, and the relative changes also, as percentages of the reference values in Equations 1 and 2 (m22c-1 and m22c-2, respectively).

Age Group	m22c	m22c-1	m22c-2	xDc(22)1	xDc(22)2	xDc(22)1%	xDc(22)2%
< 1	**31 904**	**34 762**	**37 249**	**-2 858**	**-5 345**	**-8,22 %**	**-14,35 %**
Female	13 964	14 904	16 122	-940	-2 158	-6,31 %	-13,39 %
Male	17 940	19 858	21 127	-1 918	-3 187	-9,66 %	-15,08 %
1-4	**1 923**	**1 881**	**2 077**	**42**	**-154**	**2,23 %**	**-7,41 %**
Female	736	671	684	65	52	9,69 %	7,60 %
Male	1 187	1 210	1 393	-23	-206	-1,90 %	-14,79 %
5-14	**4 674**	**4 086**	**4 323**	**588**	**351**	**14,39 %**	**8,12 %**
Female	1 510	1 378	1 553	132	-43	9,58 %	-2,77 %
Male	3 164	2 708	2 770	456	394	16,84 %	14,22 %
15-24	**65 773**	**51 324**	**55 119**	**14 449**	**10 654**	**28,15 %**	**19,33 %**
Female	16 369	12 526	13 484	3 843	2 885	30,68 %	21,40 %
Male	49 404	38 798	41 635	10 606	7 769	27,34 %	18,66 %
25-34	**143 510**	**106 589**	**107 228**	**36 921**	**36 282**	**34,64 %**	**33,84 %**
Female	41 427	31 335	31 626	10 092	9 801	32,21 %	30,99 %
Male	102 083	75 254	75 602	26 829	26 481	35,65 %	35,03 %
35-44	**213 567**	**149 601**	**143 962**	**63 966**	**69 605**	**42,76 %**	**48,35 %**
Female	73 111	52 602	51 876	20 509	21 235	38,99 %	40,93 %
Male	140 456	96 999	92 086	43 457	48 370	44,80 %	52,53 %
45-54	**378 936**	**295 773**	**312 760**	**83 163**	**66 176**	**28,12 %**	**21,16 %**
Female	140 457	113 601	122 230	26 856	18 227	23,64 %	14,91 %
Male	238 479	182 172	190 530	56 307	47 949	30,91 %	25,17 %
55-64	**852 061**	**686 034**	**683 414**	**166 027**	**168 647**	**24,20 %**	**24,68 %**
Female	328 443	268 402	268 976	60 041	59 467	22,37 %	22,11 %
Male	523 618	417 632	414 438	105 986	109 180	25,38 %	26,34 %
65-74	**1 297 690**	**1 009 880**	**976 016**	**287 810**	**321 674**	**28,50 %**	**32,96 %**
Female	544 643	428 337	417 570	116 306	127 073	27,15 %	30,43 %
Male	753 047	581 543	558 446	171 504	194 601	29,49 %	34,85 %
75-84	**1 527 855**	**1 247 039**	**1 211 460**	**280 816**	**316 395**	**22,52 %**	**26,12 %**
Female	730 408	604 010	591 544	126 398	138 864	20,93 %	23,47 %
Male	797 447	643 029	619 916	154 418	177 531	24,01 %	28,64 %
85+	**1 797 070**	**1 586 373**	**1 622 052**	**210 697**	**175 018**	**13,28 %**	**10,79 %**
Female	1 088 768	973 291	1 004 787	115 477	83 981	11,86 %	8,36 %
Male	708 302	613 082	617 265	95 220	91 037	15,53 %	14,75 %
Total	**6 314 963**	**5 173 342**	**5 155 660**	**1 141 621**	**1 159 303**	**22,07 %**	**22,49 %**

Table 2. Estimated excess mortality of the covid period in the USA, by age group and by sex. m22c is the total deaths during the covid period (from March 2020 to December 2021, included). m22c-1 is the total deaths during the 1st-prior 22-month period before the covid period (from May 2018 to February 2020, included). m22c-2 is the total deaths during the 2nd-prior 22-month period before the covid period (from July 2016 to April 2018, included). xDc(22)1 and xDc(22)2 correspond to the excess mortality in the covid period, calculated from Equation 1 and Equation 2, respectively. xDc(22)1% and xDc(22)2% correspond to the relative changes,

calculated from Equation 3 and Equation 4, respectively. ACM data were retrieved from CDC (CDC, 2022a), as described in Table 1.

One of the most surprizing results from the above calculations is that young adults were severely negatively impacted in the covid period, more so in comparative terms (percent mortality increase relative to pre-covid values) than elderly persons. This is explored further, below.

In the next section, we follow the same method to estimate the excess mortality of the covid period in the USA from a different dataset: the all-cause mortality by week (ACM/w).

3.2. USA all-cause mortality by week, by age group, 2015-2022

3.2.1. Historic trend, discontinuous break on 11 March 2020, entering the covid period

Figure 5 shows the all-cause mortality by week (ACM/w) for the USA from January 2015 to January 2022.

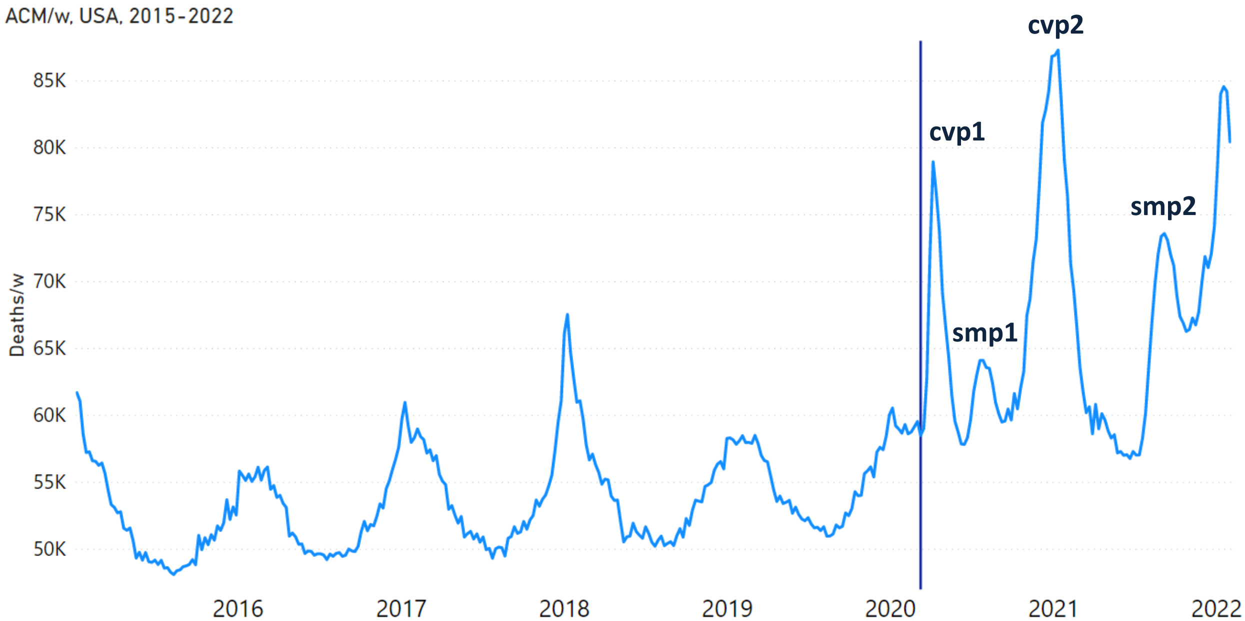


Figure 5. All-cause mortality by week in the USA from 2015 to 2022. Data are displayed from week-1 of 2015 to week-5 of 2022. The vertical dark-blue line indicates the week-11 of 2020 (week of 11 March 2020, when WHO declared a pandemic), intended to point the beginning of the covid period. The cvp1, smp1, cvp2 and smp2 features discussed in the text are indicated. Data were retrieved from CDC (CDC, 2022b), as described in Table 1.

The regular seasonal variation of mortality is seen from 2015 to early 2020, and from week-11 of 2020 (the week the WHO declared a pandemic), a new pattern of mortality (new regime of ACM by time) occurs (Figure 5, after the vertical dark-blue line). This new pattern includes the previously discussed features: cvp1, smp1, cvp2, smp2.

In the next section, we use the weekly ACM data to quantify the total excess mortality that occurred in the covid period, which includes these anomalous features.

3.2.2. Quantifying the excess mortality of the covid period, by age group

We use the ACM/w data of Figure 5 to quantify the excess deaths of the covid period "to date", compared to the historic trend, as follows.

For a given age group, we add all the weekly deaths together, for the weeks of 11 March 2020 (week-11 of 2020, start of the pandemic period; announced by the WHO on

11 March 2020) through to the latest useable week (week-5 of 2022, beginning of February 2022). This is a total for 100 weeks (the covid period “to date”). We call this total “w100c”. Then we perform a similar total for the 1st-prior 100-week period, immediately preceding the covid period, for the 100 weeks up to and including week-10 of 2020. We call this total “w100c-1”. And we do the same for the 2nd-prior 100-week period, and we call this total “w100c-2”. We cannot move back further in time with this dataset, as the “w100c-3” would be incomplete (less than a 100 weeks, with the available data).

Figure 6 shows the graph of “w100c-x” versus time, together with the ACM/w for the USA where each 100-week period has been emphasized with a different color; thus applying the same method as in producing Figure 3.

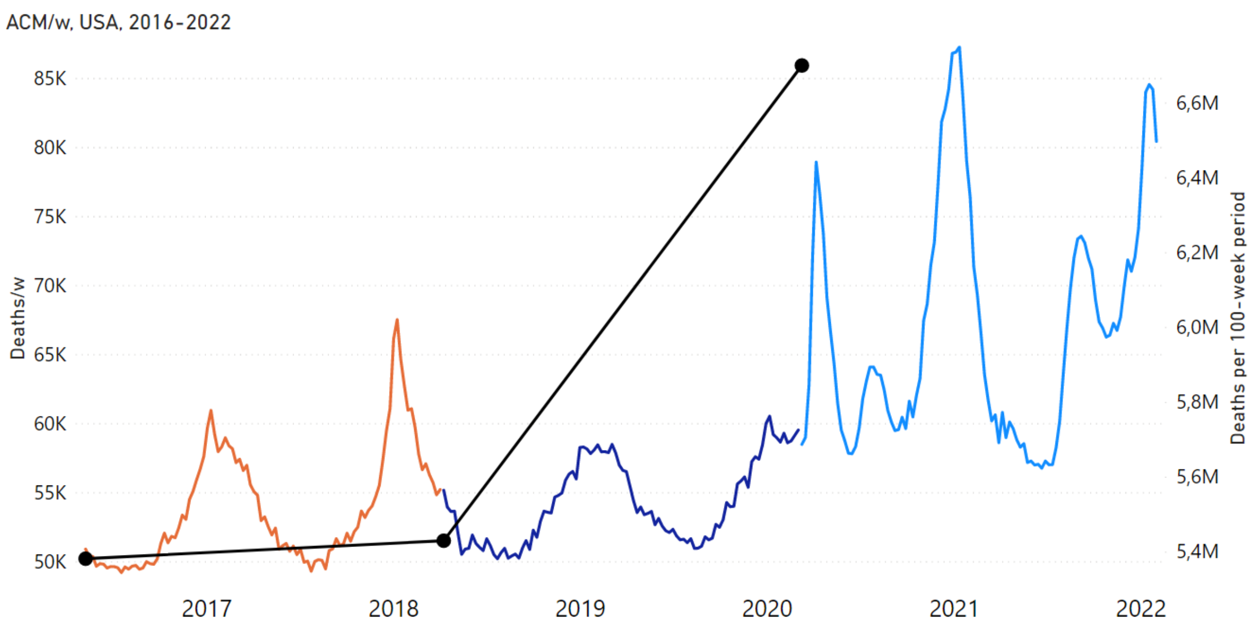


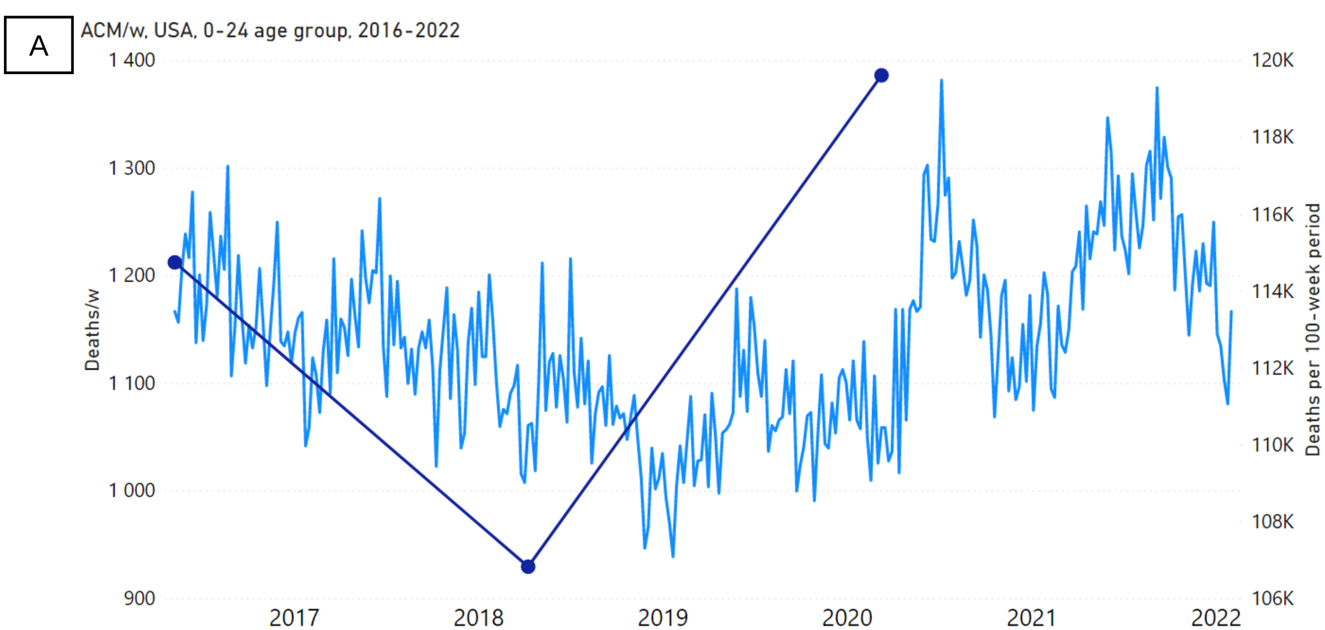
Figure 6. All-cause mortality by week (colors) and by 100-week period (black) in the USA from 2016 to 2022. Data are displayed from week-19 of 2016 to week-5 of 2022. The different colors indicate the successive 100-week periods. The light-blue color corresponds to the covid period. The dark-blue and the orange colors are in the pre-covid period. The black dots show the integrated ACM on these 100-week periods. Data were retrieved from CDC (CDC, 2022b), as described in Table 1.

Figure 6, based on more than 7 years of data, here time-resolved by week, again (like Figure 3) dramatically illustrates the sudden change in regime of ACM by time, both in magnitude of time-integrated ACM and in seasonal behaviour of ACM by time, occurring as soon as the WHO on 11 March 2020 announced a pandemic. In addition, the covid-period regime of ACM by time is characterized by large (and unprecedented in the historic record) heterogeneity by state of ACM, which is not shown in such a figure for the whole USA, but which can be appreciated in the ACM/w by state graphs of Appendix A.

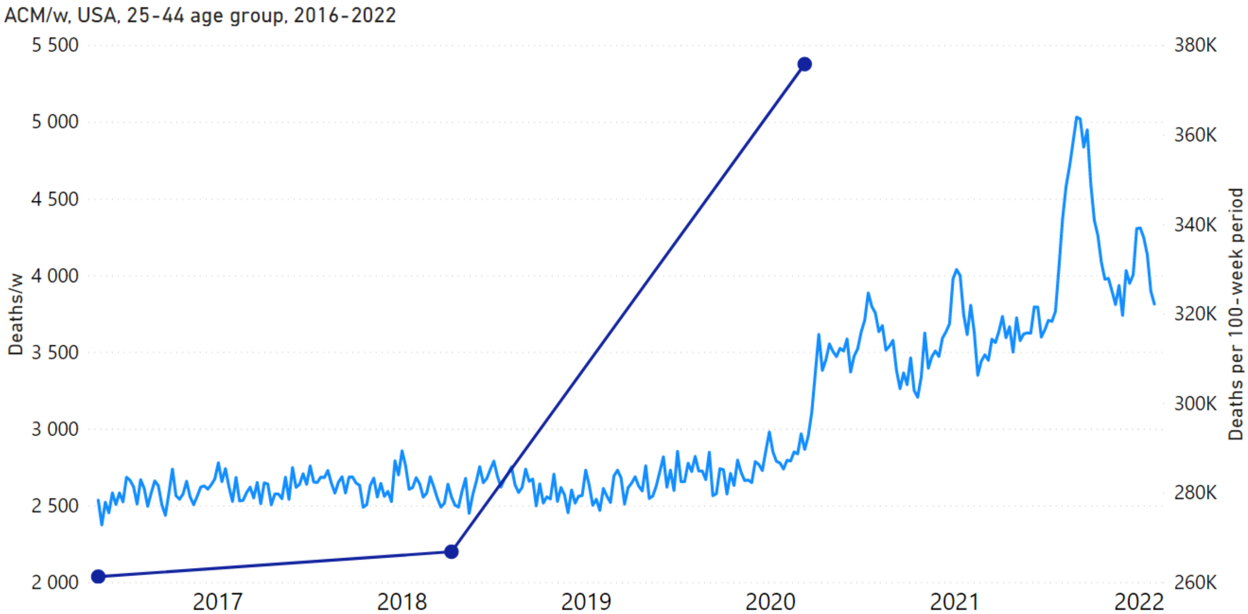
For the whole USA (all states and all ages together), the increase in ACM between the pre-covid and the covid period is close to 25% (Figure 6).

The mortality data (Figure 6) can be resolved by age group, which is shown, as follows, in Figure 7.

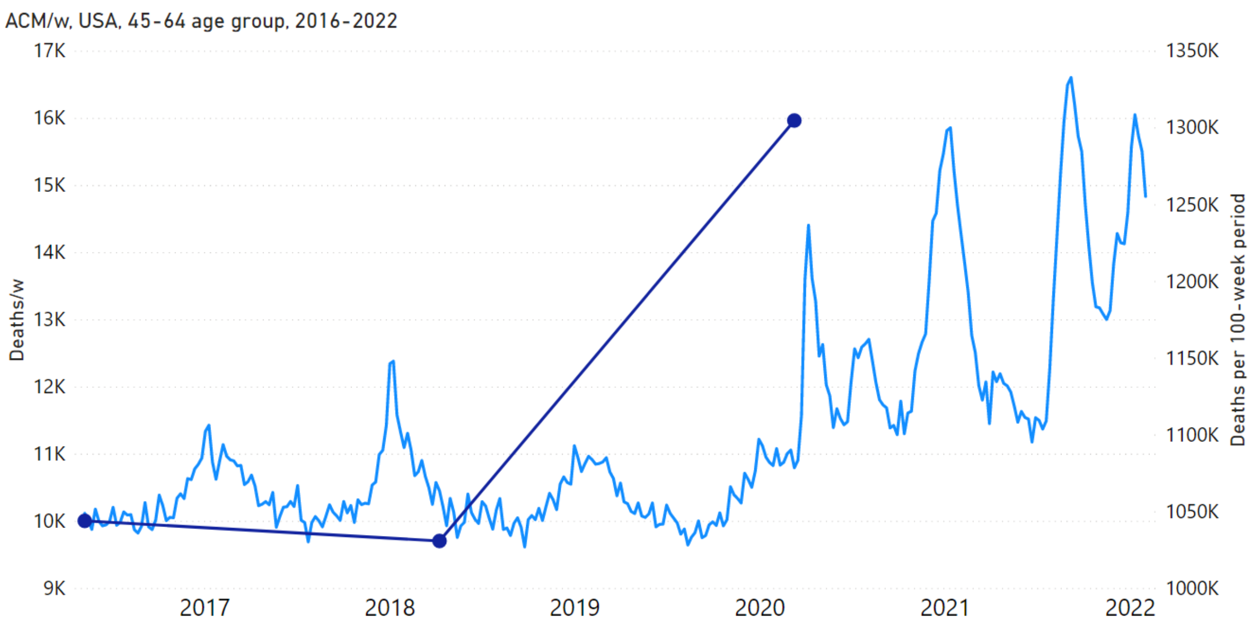
Figure 7. All-cause mortality by week (light-blue) and by 100-week period (dark-blue) in the USA from 2016 to 2022, for each of the age groups. Data are displayed from week-19 of 2016 to week-5 of 2022. Panels below: (A) for the 0-24 years age group; (B) for the 25-44 years age group; (C) for the 45-64 years age group; (D) for the 65-74 years age group; (E) for the 75-84 years age group; (F) for the 85+ years age group. Data were retrieved from CDC (CDC, 2022b), as described in Table 1.



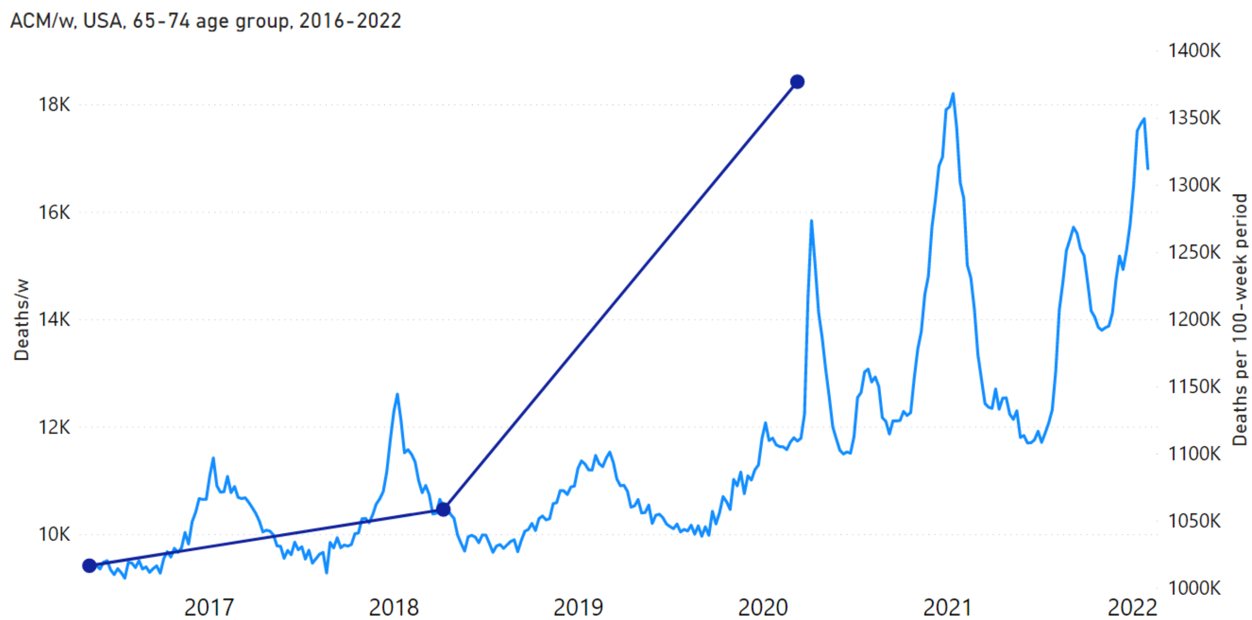
B


ACM/w, USA, 25-44 age group, 2016-2022
Deaths/w
5 500
5 000
4 500
4 000
3 500
3 000
2 500
2 000
2017
2018
2019
2020
2021
2022
380K
360K
340K
320K
300K
280K
260K
Deaths per 100-week period

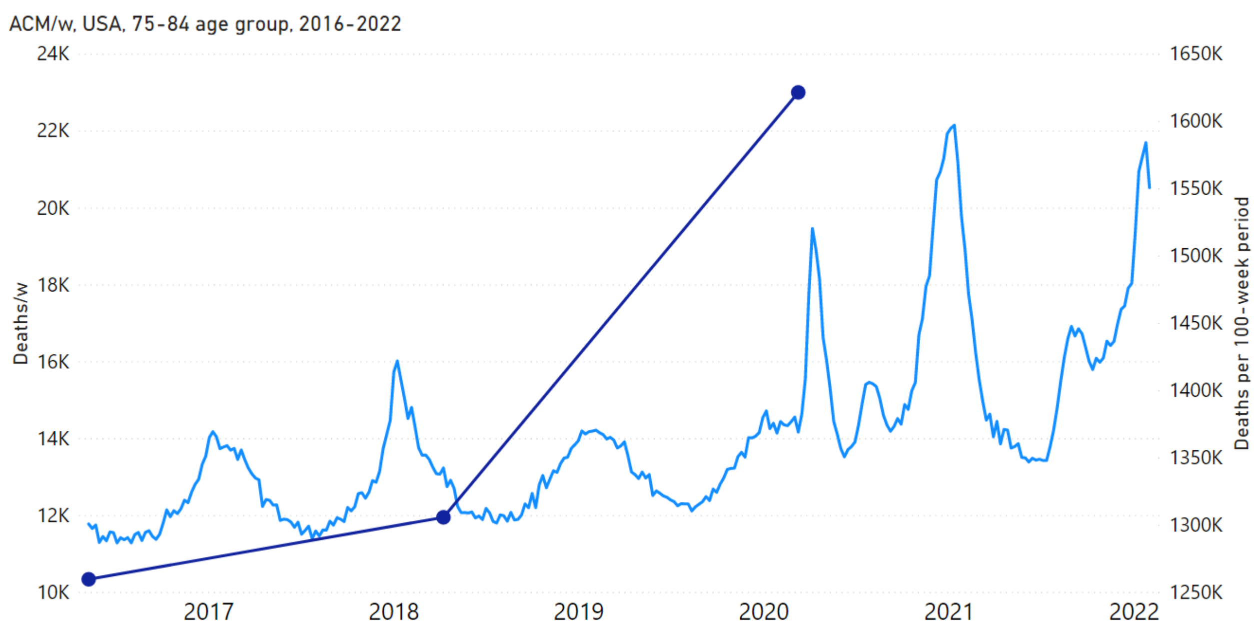
C


ACM/w, USA, 45-64 age group, 2016-2022
Deaths/w
17K
16K
15K
14K
13K
12K
11K
10K
9K
2017
2018
2019
2020
2021
2022
1350K
1300K
1250K
1200K
1150K
1100K
1050K
1000K
Deaths per 100-week period

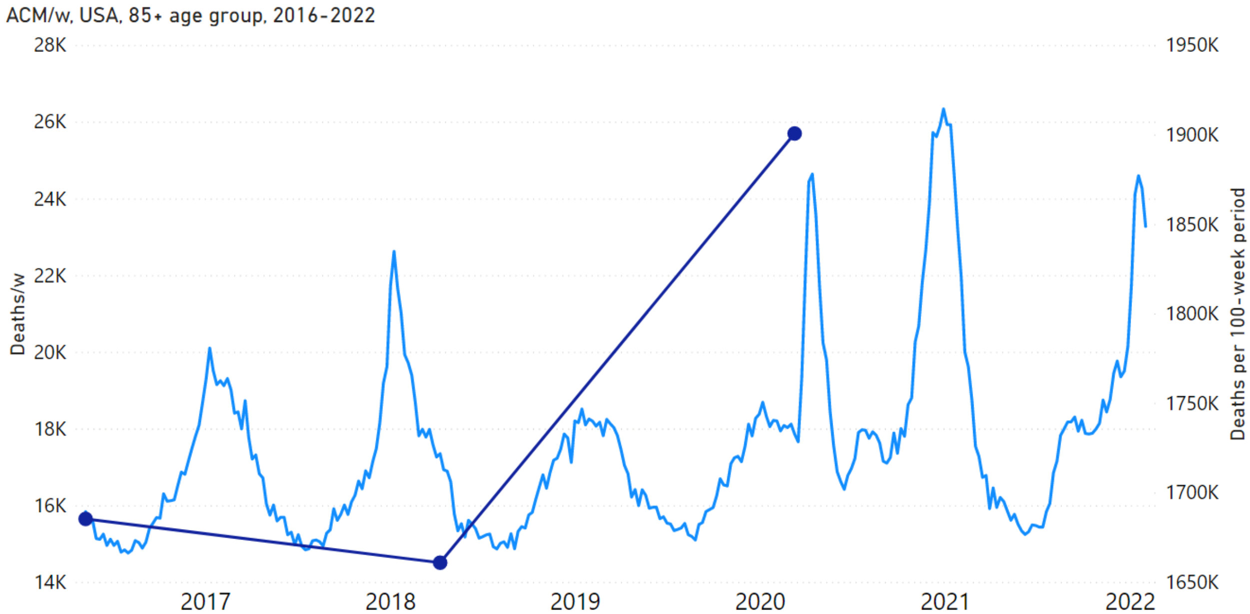
D


ACM/w, USA, 65-74 age group, 2016-2022
18K
16K
14K
12K
10K
Deaths/w
1400K
1350K
1300K
1250K
1200K
1150K
1100K
1050K
1000K
Deaths per 100-week period
2017
2018
2019
2020
2021
2022

E


ACM/w, USA, 75-84 age group, 2016-2022
24K
22K
20K
18K
16K
14K
12K
10K
Deaths/w
1650K
1600K
1550K
1500K
1450K
1400K
1350K
1300K
1250K
Deaths per 100-week period
2017
2018
2019
2020
2021
2022

F



Except for the younger age group (the 0-24 year-olds, Figure 7A), the integrated mortality of the covid period is much larger than in any of the two previous 100-week periods (Figure 7B, C, D, E, F). These results are comparable to those illustrated in Figure 4.

It is interesting to note that the sudden rise in ACM, immediately following the WHO's 11 March 2020 declaration of a pandemic, which we have discussed in several previous articles (Rancourt, 2020; Rancourt, Baudin and Mercier, 2020, 2021a, 2021b), occurs in all the age groups for the whole USA (Figure 7; and see Figure 4), not solely in the most elderly populations as reports of severe COVID-19 morbidity might lead one to conclude (e.g., Elo *et al.*, 2022; Sorensen *et al.*, 2022). This, in itself, suggests that the covid-period deaths are not predominantly explained by the postulated SARS-CoV-2 pathogen.

Next, for a given age group, we calculate the excess deaths of the covid period "to date" using our simplest assumption from above. We take the "excess deaths of the covid period" to mean the (all-cause) deaths above the deaths that would have occurred if the same circumstances would have prevailed during the covid period as prevailed in the 1st-prior 100-week period, immediately preceding the covid period. Under this

assumption, the excess deaths of the covid period, due to everything different or extraordinary that occurred or was imposed during the covid period, for a given age group and state, is:

$$xDc(100)1 = w100c - w100c\text{-}1 \qquad (5)$$

In the second assumption, we take the "excess deaths of the covid period" to mean the (all-cause) deaths above the deaths that would have occurred if the same circumstances would have prevailed during the covid period as prevailed in the 2nd-prior 100-week period, the period preceding the 1st-prior 100-week period before the covid period. Under this assumption, the excess deaths of the covid period, due to everything different or extraordinary that occurred or was imposed during the covid period, for a given age group and state, is:

$$xDc(100)2 = w100c - w100c\text{-}2 \qquad (6)$$

As with Equations 1 and 2 above, these formulas (Equations 5 and 6) are justified because w100c-1 and w100c-2 are different and fair estimates of what mortality would have been in the 100-week covid period if the events associated with the declared pandemic had not occurred. In other words, w100c-1 and w100c-2 are fair historical projected values of what the covid-period mortality "would have been". Judging from Figure 6 and Figure 7, there would be little benefit from applying a more mathematically sophisticated extrapolation method, while using both reference values allows one to estimate the uncertainty in our determinations of excess mortality for the covid period.

The relative magnitudes of the covid-period extra deaths above the historic trend are:

$$xDc(100)1\% = xDc(100)1 \;/\; w100c\text{-}1, \text{ expressed as a percentage,} \qquad (7)$$

and

$$xDc(100)2\% = xDc(100)2 \;/\; w100c\text{-}2, \text{ expressed as a percentage,} \qquad (8)$$

Table 3 contains the thus calculated covid-period excess mortality, for each age group for the USA, and for the entire USA (“Total”), using each assumption described above, and the relative changes also, as percentages of the reference values in Equations 5 and 6 (w100c-1 and w100c-2, respectively).

Age Group	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
0-24	119 621	106 833	114 753	12 788	4 868	11,97 %	4,24 %
25-44	375 881	266 917	261 359	108 964	114 522	40,82 %	43,82 %
45-64	1 304 887	1 031 015	1 044 091	273 872	260 796	26,56 %	24,98 %
65-74	1 377 235	1 058 708	1 016 822	318 527	360 413	30,09 %	35,45 %
75-84	1 621 693	1 305 924	1 259 881	315 769	361 812	24,18 %	28,72 %
85+	1 900 921	1 661 094	1 685 547	239 827	215 374	14,44 %	12,78 %
Total	**6 700 238**	**5 430 491**	**5 382 453**	**1 269 747**	**1 317 785**	**23,38 %**	**24,48 %**

Table 3. Estimated excess mortality of the covid period in the USA, by age group. w100c is the total deaths during the covid period (from week-11 of 2020 to week-5 of 2022, included). w100c-1 is the total deaths during the 1st-prior 100-week period before the covid period (from week-15 of 2018 to week-10 of 2020, included). w100c-2 is the total deaths during the 2nd-prior 100-week period before the covid period (from week-19 of 2016 to week-14 of 2018, included). xDc(100)1 and xDc(100)2 correspond to the excess mortality in the covid period, calculated from Equation 5 and Equation 6, respectively. xDc(100)1% and xDc(100)2% correspond to the relative changes, calculated from Equation 7 and Equation 8, respectively. ACM data were retrieved from CDC (CDC, 2022b), as described in Table 1.

Equivalents to Table 3 for each of the states of the USA can be found in Appendix A.

Not surprisingly, we find the same results as with the ACM/m data, where young adults were relatively more impacted in the covid period than elderly persons.

In the next section, we explore the excess mortality of the covid period at the state level.

3.2.3. Excess mortality of the covid period, by state

Figure 8 shows USA maps of the state-wise values of the covid-period excess mortality (xDc(100)1), as relative changes in percentage of the pre-covid period mortality (xDc(100)1%) (Panel A), and xDc(100)1 per state population (Panel B), for comparison.

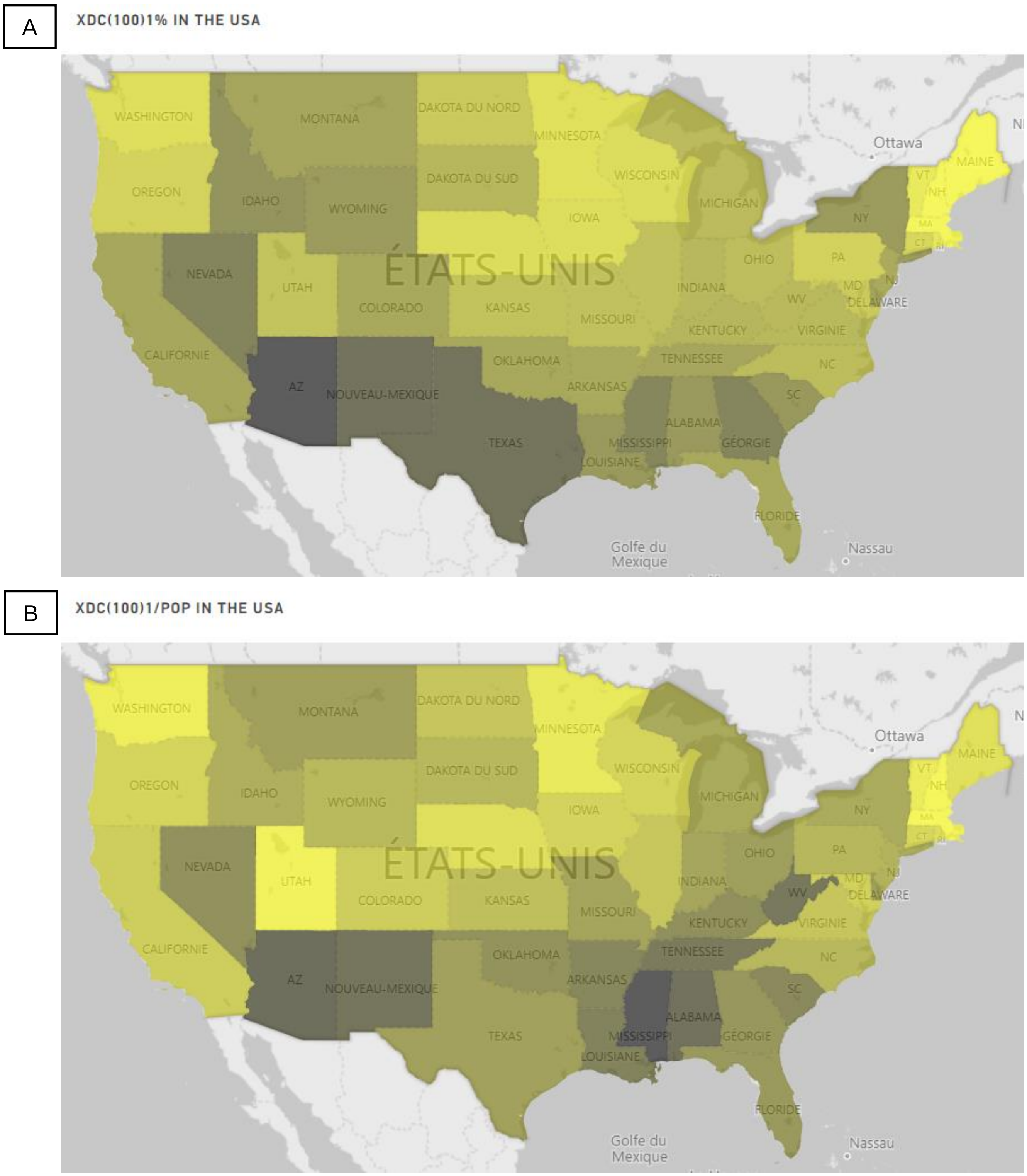


Figure 8. Maps of the excess mortality of the covid period in the USA, as percentages of the pre-covid period mortality (panel A) and as normalized by state population (panel B). Alaska and Hawaii are excluded. The darker the color (black), the more intense is the relative change. ACM data were retrieved from CDC (CDC, 2022b) and population data were retrieved

from US Census Bureau (US Census Bureau, 2021), as described in Table 1. xDc(100)1 and xDc(100)1% are calculated from Equation 5 and Equation 7, respectively.

These maps (Figure 8) can be compared to the maps of poverty and obesity shown in Appendix B; and to the maps from Rancourt et al. (Rancourt, Baudin and Mercier, 2021b) of life expectancy (their Figure 38a), antibiotic prescriptions (their Figure 38b), average climatic temperature (their Figure 22), intensity of the smp1 mortality (their Figure 16), intensity of the cvp1 mortality (their Figure 15). Some of these comparisons are discussed further below.

Generally, high 100-week covid-period mortality per capita or per baseline mortality occurs in the Southern states, and in the hottest climatic state of Arizona. This is similar to what we have reported previously for summer-season covid mortality (Rancourt, Baudin and Mercier, 2021b). Below we show that state-wise covid-period mortality is very strongly correlated (r = +0.86) to state-wise poverty, and also correlated to median household income, obesity, disability, and government subsidy programs; which in turn are known to be correlated to each other and to diabetes prevalence, life expectancy, and antibiotic prescriptions. All of this is consistent with the geographical pattern shown in Figure 8.

Figure 9 shows the xDc(100)1% (Equation 7) values from Table 3 by age group, for the whole USA (Panel A), and for the ten most populous states (Panel B), ordered from the most populous to the less populous (US Census Bureau, 2022a): California, Texas, Florida, New York, Pennsylvania, Illinois, Ohio, Georgia, North Carolina and Michigan. The horizontal dashed line represents the value for the whole USA (all ages and all states).

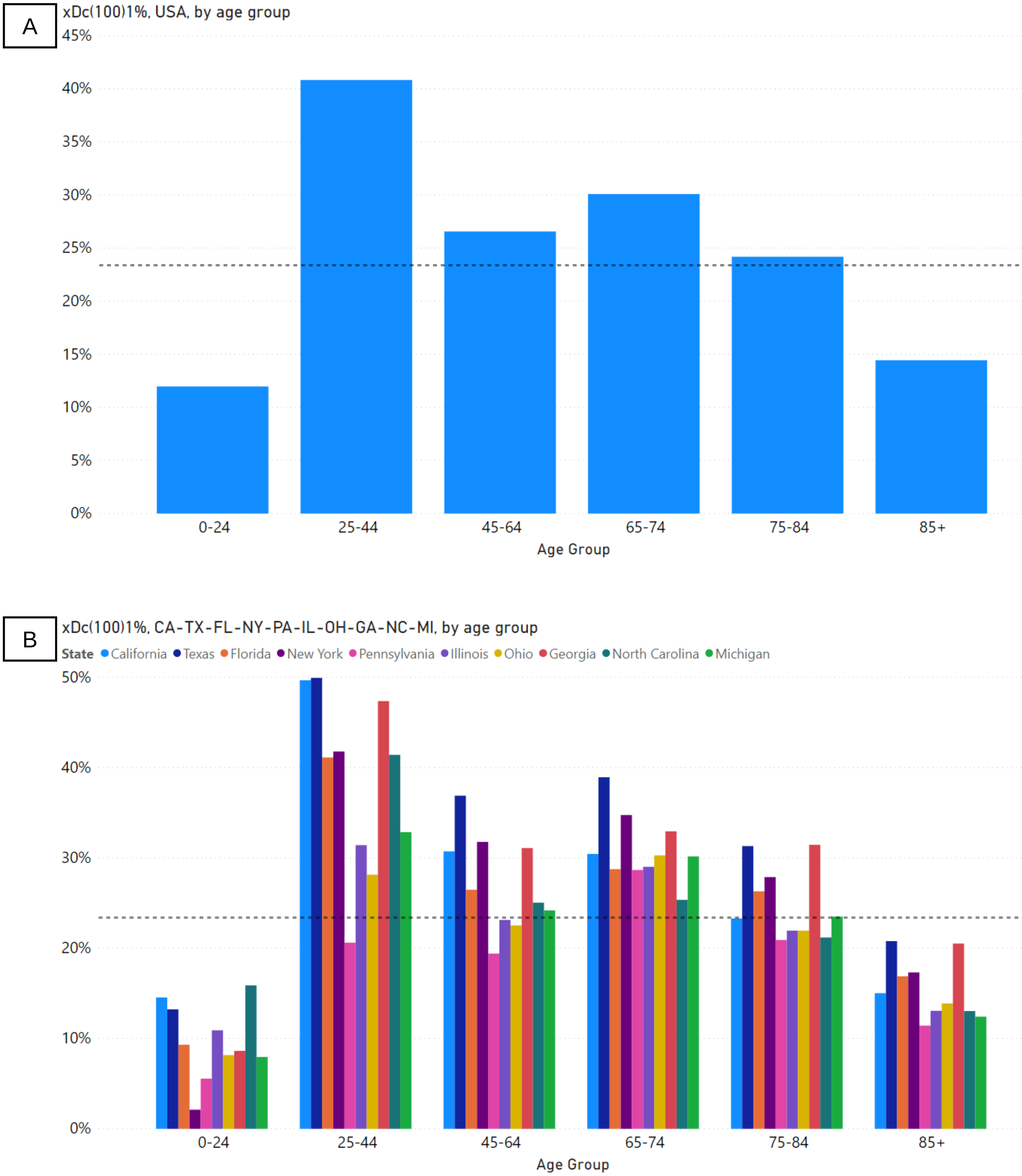


Figure 9. Excess mortality of the covid period in the USA (panel A) and in the ten most populous states of the USA (from left to right in each band: California, Texas, Florida, New York, Pennsylvania, Illinois, Ohio, Georgia, North Carolina, Michigan) (panel B), as percentages of the pre-covid period mortality, by age group. The constant dashed line

represents the value for the whole USA. ACM data were retrieved from CDC (CDC, 2022b), as described in Table 1. xDc(100)1% is calculated from Equation 7.

Figure 9 illustrates one of the most striking features of mortality in the covid period: The relative covid-period excess mortality (covid-period fatality risk ratio, relative to pre-covid mortality) is broadly distributed to all age groups and is not exponential or near-exponential with age as determined for viral respiratory diseases, including COVID-19, when these are the verified dominant cause of death.

Indeed, we note that all age groups were significantly differentially affected in the covid period, which is inconsistent with the reported infection fatality ratios (morbidity) that generally increase exponentially with age, as is also the case for many chronic diseases and for all-cause mortality risk itself (e.g., Richmond *et al.*, 2021; Elo *et al.*, 2022; Sorensen *et al.*, 2022). Again, this suggests that the covid-period deaths are not predominantly explained by the postulated SARS-CoV-2 pathogen. Rather, risk of death in the covid period appears to result from distributed aggression against vulnerable populations in all the age groups, not predominantly (or exponentially) the elderly.

We see from Figure 9 that young adults (25-44 years) were particularly devastated by the events and conditions of the covid period. It is not unreasonable to postulate that this age group would have been most impacted by the large-scale life-changing economic and job-loss changes that occurred in the covid period, or that this age group would have been most devastated by social isolation and institutional abandonment for those who are mentally disabled or otherwise dependent on a fragile social support network.

Next, we examine whether any impact of the mass and age-distributed USA vaccination campaign can be detected and quantified.

3.3. Time and age-group variations of mortality during the covid period, and relation to implementation of the vaccination campaign

3.3.1. All-cause mortality by week and vaccination delivery by week, by age group, 2019-2022

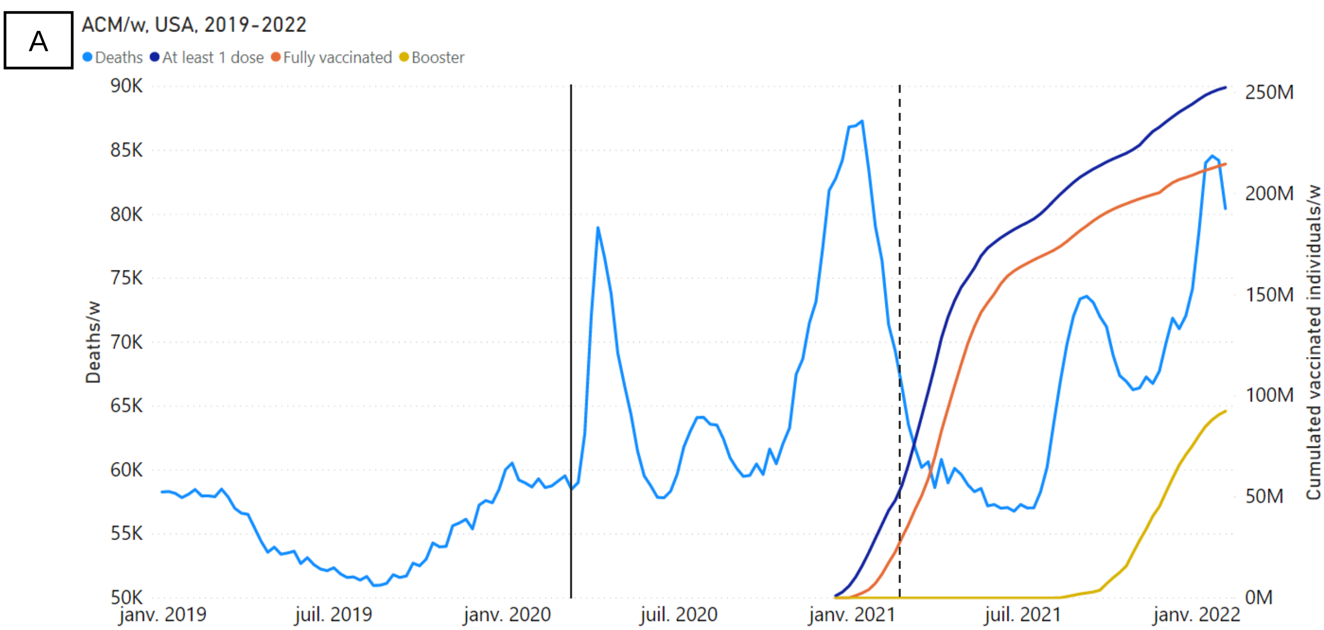
In our previous article about ACM in the USA (Rancourt, Baudin and Mercier, 2021b), we stated the following about the vaccination campaign:

> "Readers who would be tempted to ascribe the downturn in the cvp2 peak to the vaccination campaign should note that the downturn coincides with the expected seasonal downturn of every seasonal winter maximum that has ever been observed by epidemiologists in the last century or more.
>
> More importantly, the largely completed vaccination campaign did not prevent a second surge of summer deaths (2021, "smp2") (Figure 31). The mortality in the said second surge appears to be comparable to or more than the mortality for summer-2020. Furthermore, the COVID-19-assigned deaths (CDC, 2021a) are significantly greater in number in summer-2021 than in summer-2020 (Figure 34), and, unlike at any other time in the COVID-era, account for virtually all the excess (above-SB) deaths, in the summer-2021 feature (smp2) (Figure 34), following the vaccination campaign.
>
> There is no sign in the ACM/w that the vaccination campaign has had any positive effect. However, given that the vaccination campaign starts well after the 2020 summer and essentially ends mid-summer-2021 prior to the start of the smp2 feature, given that the 2021 excess (above-SB) summer deaths (smp2) occur in significantly younger individuals than the excess summer-2020 deaths, and given that the smp2 feature is significantly larger than the smp1 feature for the said younger individuals (35-54 years, Figures 33d and 33e; and 55-64 years, Figure 33f, to a lesser degree), it is possible that vaccination made 35-54 year olds and others more vulnerable to death, especially summer death in disadvantaged individuals in hot-climate states (Montgomery et al., 2021) (Simone et al., 2021)."

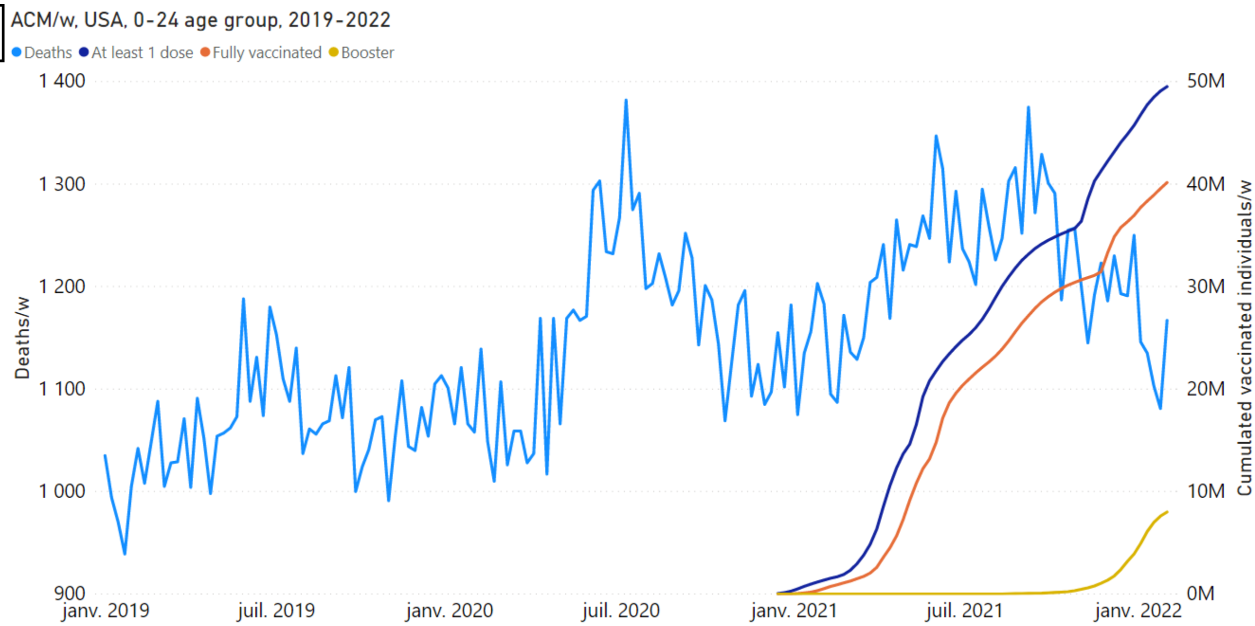
Here, we examine this question again, *via* the time and age-group variations in structure of the ACM/w (Figure 5 and Figure 7) in the covid period, using the most up-to-date consolidated data.

Figure 10 shows the all-cause mortality by week (ACM/w) for the USA from January 2019 through January 2022, together with vaccination data, for all the available age groups.

Figure 10. All-cause mortality by week (light-blue), cumulated number of people with at least one dose of vaccine (dark-blue), cumulated number of fully vaccinated people (orange) and cumulated number of people with a booster dose (yellow) by week in the USA from 2019 to 2022, for all and each of the age groups. Data are displayed from week-1 of 2019 to week-5 of 2022. The vertical solid line indicates week-11 of 2020 (week of 11 March 2020, when WHO declared a pandemic), indicating the beginning of the covid period. The vertical dashed line indicates week-8 of 2021, dividing the covid period into two periods of 50 weeks each: the pre-vaccination period (before the dashed line) and the vaccination period (after the dashed line). Panels below: (A) for all ages; (B) for the 0-24 years age group; (C) for the 25-44 years age group; (D) for the 45-64 years age group; (E) for the 65-74 years age group; (F) for the 75+ years age group. Data were retrieved from CDC (CDC, 2022b, 2022c), as described in Table 1.

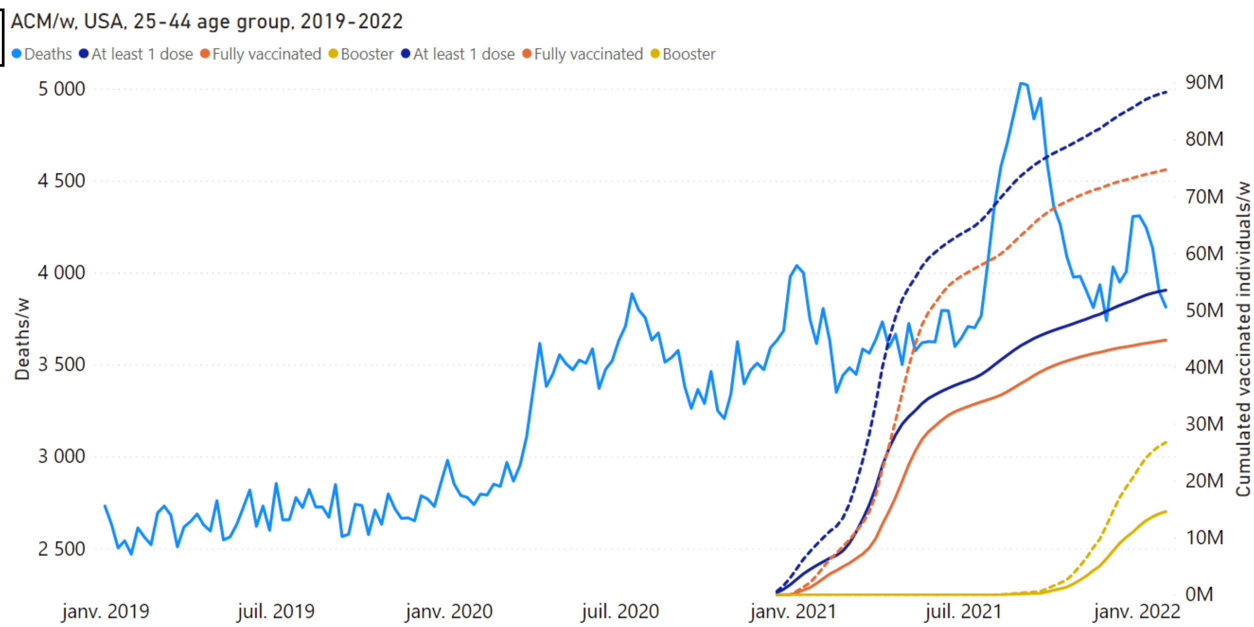


B



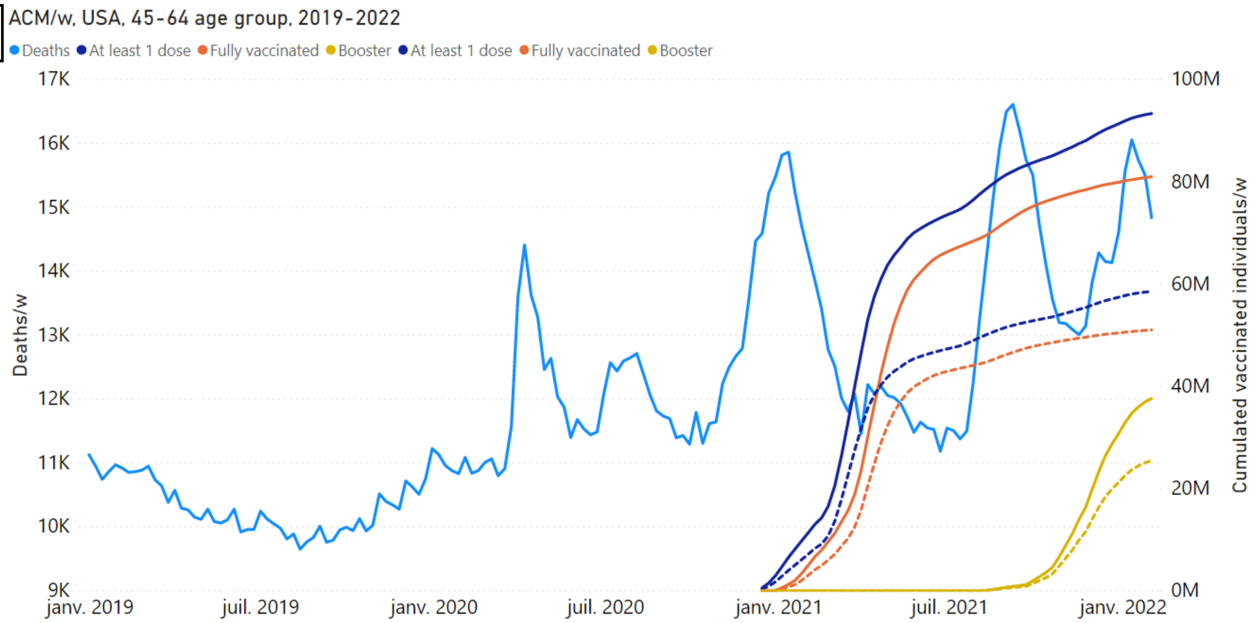
The booster data for this age group only concern people aged 12 years and older.

C



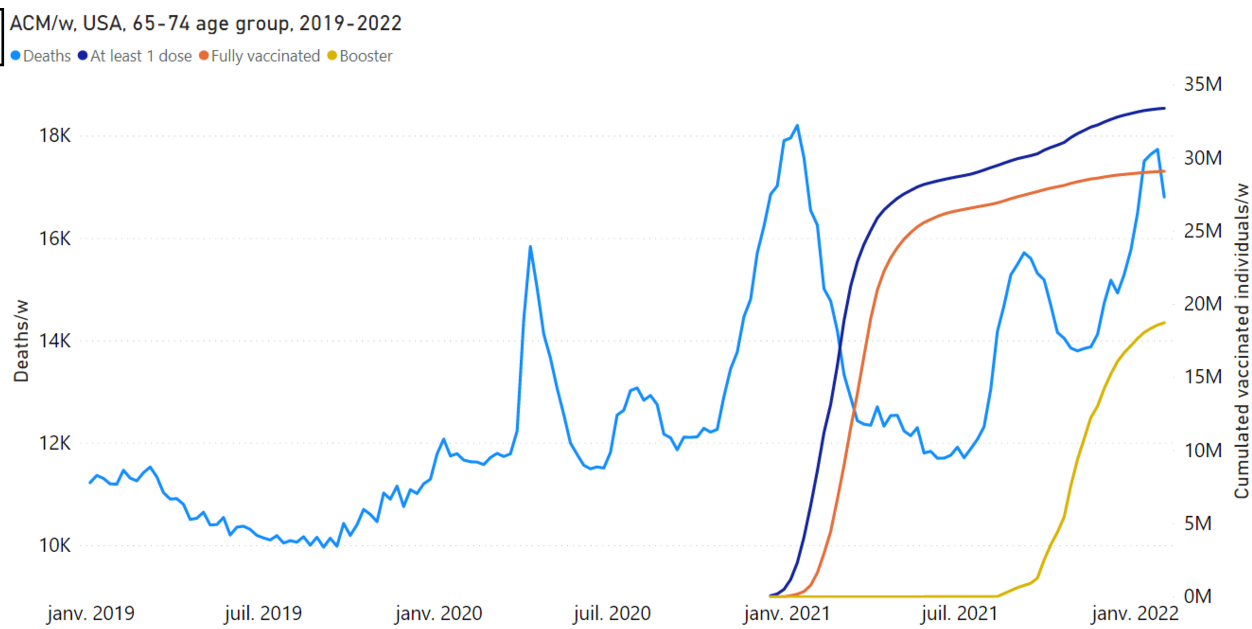
For the vaccination data of this age group, the solid lines are for the 25-39 year olds and the dashed lines are for the 25-49 year olds. That is because the available age groups for the mortality data don't exactly match the available age groups for the vaccination data.

D



For the vaccination data of this age group, the solid lines are for the 40-64 year olds and the dashed lines are for the 50-64 year olds. That is because the available age groups for the mortality data don't exactly match the available age groups for the vaccination data.

E



F

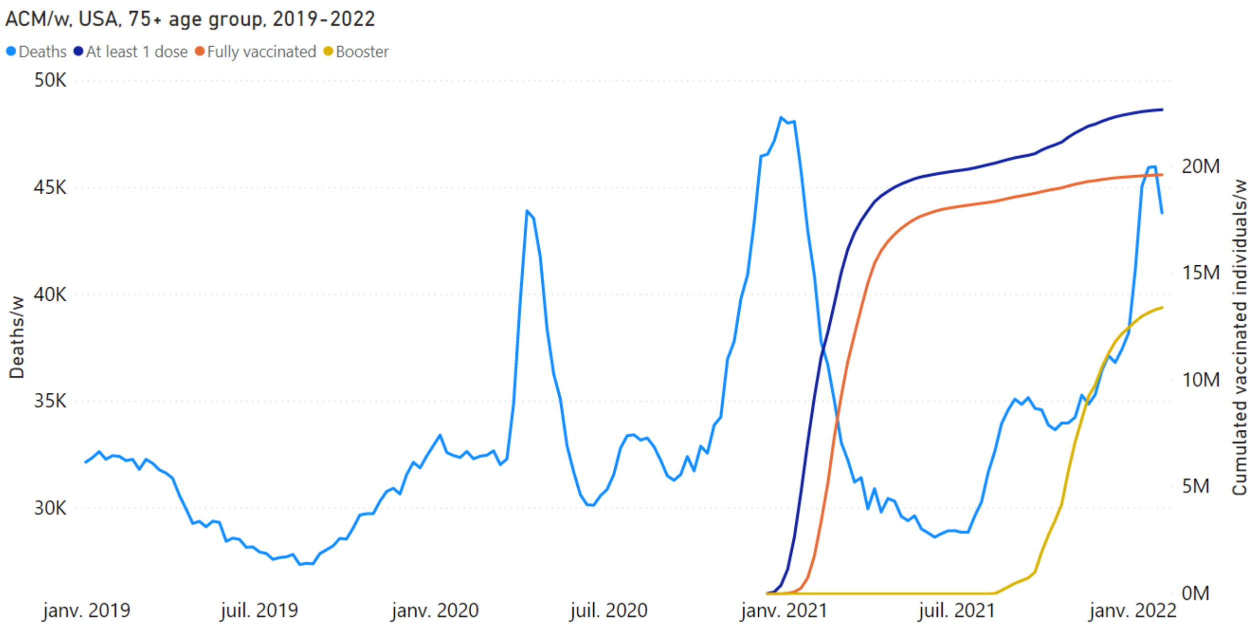


Figure 10 is a key figure in the present article because it allows an investigation of whether accelerations of vaccine delivery are synchronous or near-synchronous with surges (vaccine-induced death) or subsequent drops (vaccine-induced protection against death) in ACM, for all ages (Figure 10A) and by age group (Figure 10 B, C, D, E, F). In this regard, we make the following observations.

- First, one might be tempted to mechanistically associate the initial and most important surge in 1st-dose vaccine delivery with the large drop in mortality that for several age groups occurs at about the same time (in March-2021 for all ages, Figure 10A). This is incorrect for the following reasons:
  - The drop in mortality is expected from purely seasonal considerations: high mortality in the winter always drops eventually.
  - The cvp1 at the end of the winter occurring in the pre-vaccination period of the covid period saw an eventual large decrease, months before the start of the vaccination campaign.
  - There is an increase in ACM in the 0-24 years age group, rather than a decrease (Figure 10B), and similarly for the 25-44 years age group (Figure 10C). The vaccine would need to be harmful or beneficial regarding death, depending on the age group.

- For the 45-64, 65-74 and 75+ years age groups, the 2020-2021 winter peak in ACM occurs in the same way even though the vaccine-delivery upsurge is at different times, because the most elderly were vaccinated first (Figure 10D, E, F). The vaccine's life-saving properties would need to be strongly dependent on age for these ages.

- Second, it is clear that the prominent late-summer-2021 peak in ACM (all ages, and all age groups except 0-24 years) is far in excess of any proportionate increase in vaccination-dose delivery. The said late-summer-2021 peak occurs in a period during which the cumulative vaccine dose delivery is essentially regular, without a large fractional step-wise increase.
- Third, the latter observation notwithstanding, there is nonetheless a modest but statistically significant stepwise increase in 1st-dose vaccine delivery, which is synchronous with the late-summer-2021 peak in ACM, visible for all ages and for the 25-44 and 45-64 years age groups (Figure 10A, C, D). This temporal association is prominent in the data for many specific states (e.g., Figure 11), and cannot easily be dismissed. It is discussed below.
- While the second and third bullet points above appear to be contradictory, they are not. On the one hand (second bullet point), neither large increases in ACM (upsurge of the late-summer-2021 peak) nor large decreases in ACM (drop in ACM ending the late-summer-2021 peak) can be interpreted as proportionately driven by vaccine adverse effects, while on the other hand (third bullet point), a modest stepwise upsurge in cumulative vaccine dose delivery may be causally associated with a peak in ACM if the said stepwise upsurge includes increased capture of immunocompromised residents. The two propositions (second and third bullet points) and their implications are simultaneously possible because the number of delivered vaccine doses is large compared to the number of excess deaths (the per-dose fatality toxicity ratio of the vaccine is much smaller than 1), as discussed more below.
- Fourth, one might be tempted to mechanistically associate the increase in cumulative booster-dose delivery with irregular increases in ACM in the late stage of the covid period. This is incorrect for the following reasons:

- The apparent association is confounded by the 2021-2022 winter increase. Every winter, including during the covid period, has always had increased ACM, in the entire recorded history of mid-latitude countries and jurisdictions.
- Booster and concomitant first-series dose increases have an apparent insignificant effect on ACM in the 0-24 years age group, and cause a decrease if anything in the winter 2021-2022 season (Figure 10B).
- Boosters cause no special increase in ACM in the 25-44 years age group (Figure 10C), which is the age group with the largest vaccination-period relative increase in integrated ACM (see below).
- The 2021-2022 winter peaks in all the >24 years age groups have their maxima at a time when the cumulative booster-dose delivery has plateaued, after its period of most rapid increase (Figure 10A, C, D, E, F).

Data by age group shown in Figure 10 were only available at the national level. In the next section, we look at vaccination data at the state level, with less defined age groups.

3.3.2. All-cause mortality by week and vaccination delivery by week, by state, 2019-2022

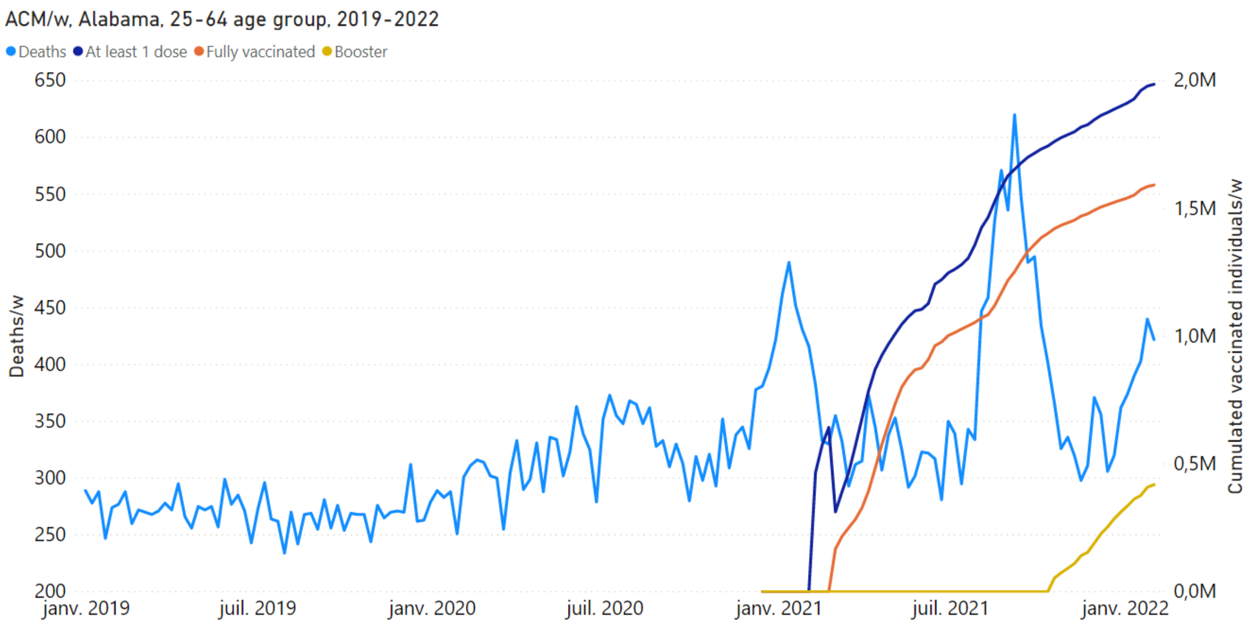
Vaccination delivery by week data is available at the state level for the 18+ and the 65+ age groups (CDC, 2022d). By subtracting the data for the 65+ age group from the data for the 18+ age group, we can calculate data for the 18-64 age group.

Figure 11 shows the all-cause mortality by week (ACM/w) for some states of the USA from January 2019 through January 2022, together with vaccination data, for the 25-64 years or the 65+ years age groups.

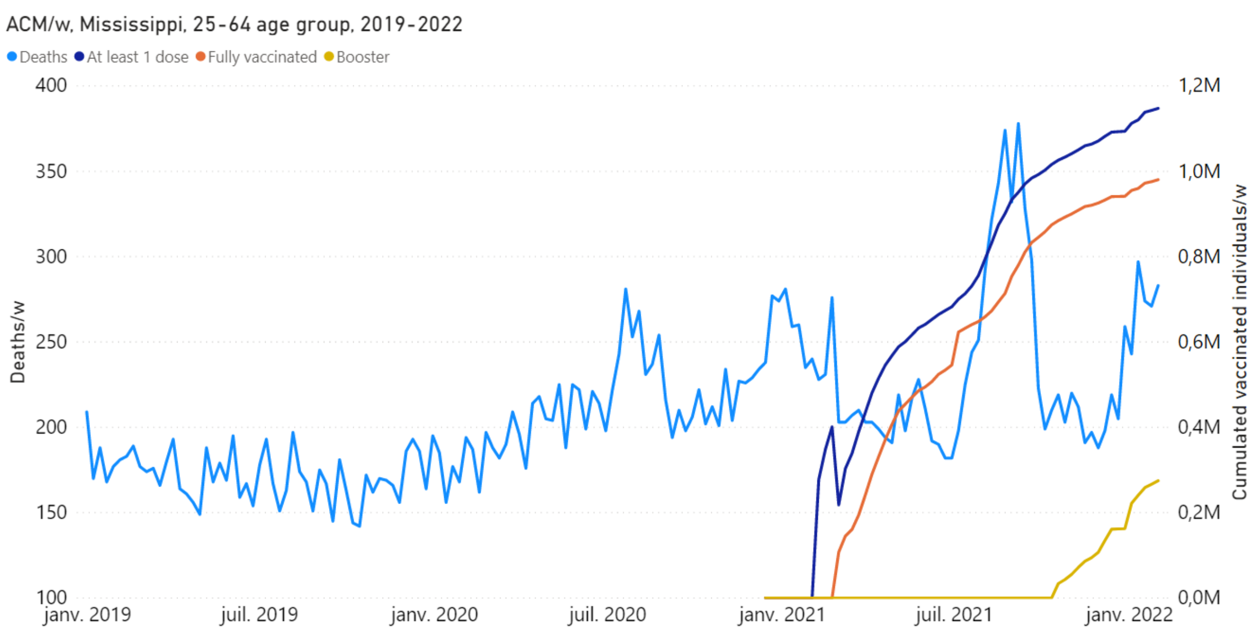
Figure 11. All-cause mortality by week (light-blue), cumulated number of people with at least one dose of vaccine (dark-blue), cumulated number of fully vaccinated people (orange) and cumulated number of people with a booster dose (yellow) by week from 2019 to 2022, and by age group for some states. Data are displayed from week-1 of 2019 to week-5 of 2022. Panels below: (A) Alabama, 25-64 years age group; (B) Mississippi, 25-64

years age group; (C) Georgia, 25-64 years age group; (D) Florida, 25-64 years age group; (E) Louisiana, 25-64 years age group; (F) Louisiana, 65+ years age group; (G) Michigan, 25-64 years age group; (H) Michigan, 65+ years age group. For the 25-64 years age group graphs, the vaccination data is for the 18-64 years age group; because the available age groups for the mortality data do not exactly match the available age groups for the vaccination data. The discontinuous breaks in cumulative number of vaccinated individuals are artifacts. Data were retrieved from CDC (CDC, 2022b, 2022d), as described in Table 1.

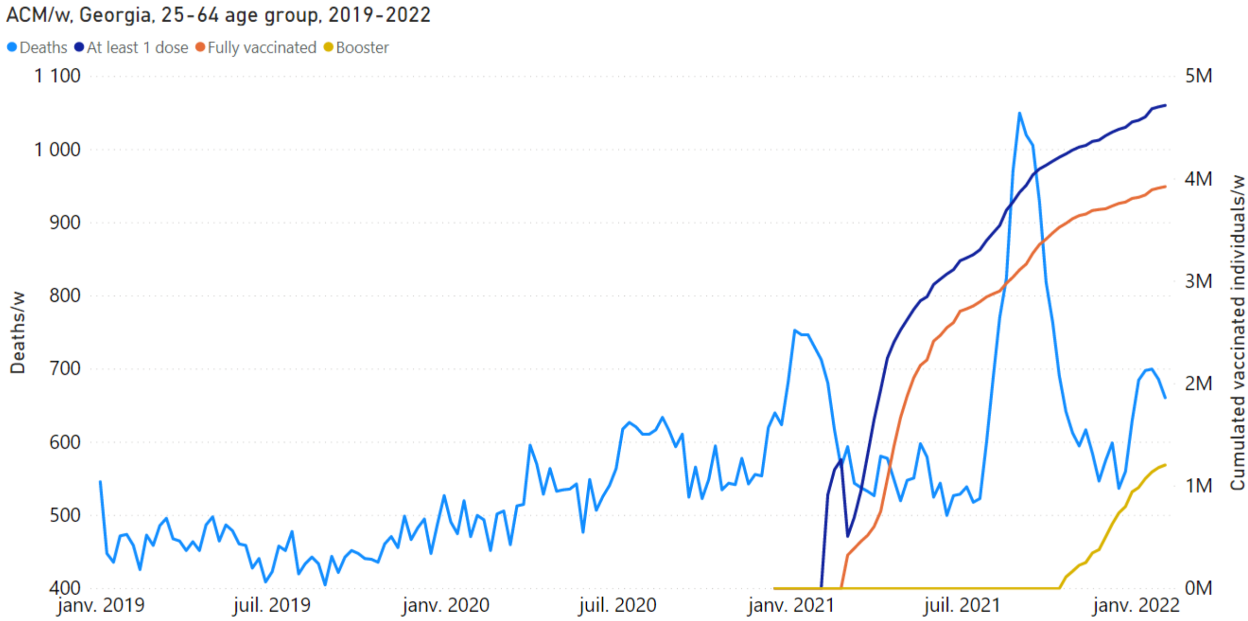
A



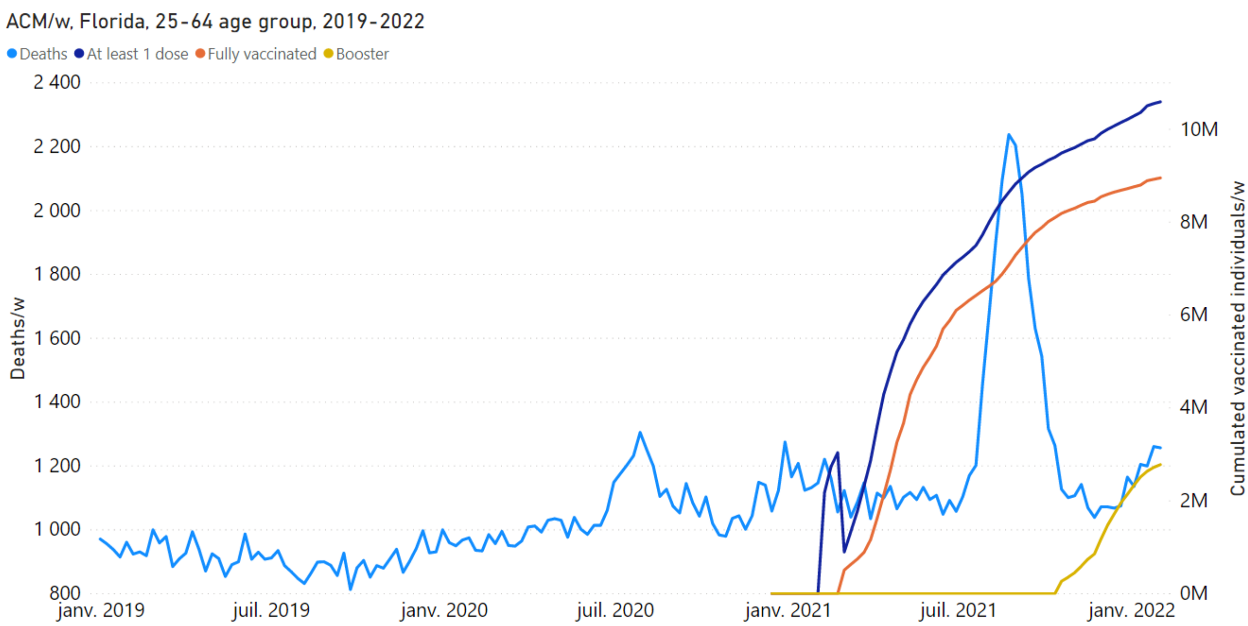
B



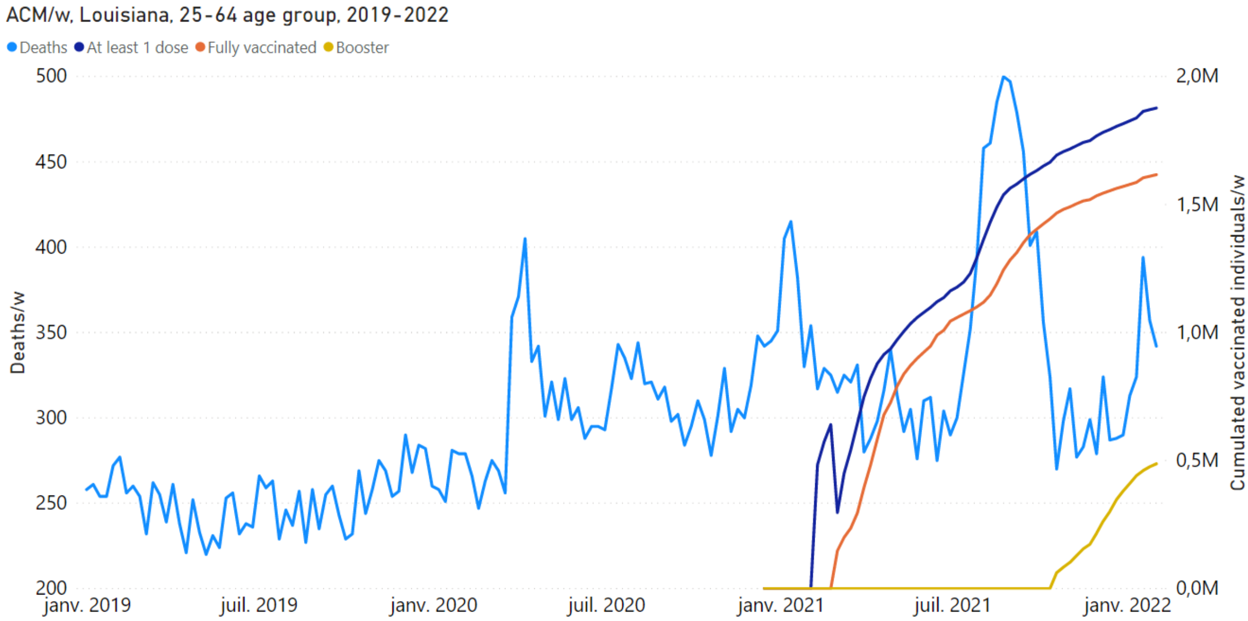
C


ACM/w, Georgia, 25-64 age group, 2019-2022
Deaths At least 1 dose Fully vaccinated Booster
1 100
1 000
900
800
700
600
500
400
Deaths/w
5M
4M
3M
2M
1M
0M
Cumulated vaccinated individuals/w
janv. 2019
juil. 2019
janv. 2020
juil. 2020
janv. 2021
juil. 2021
janv. 2022

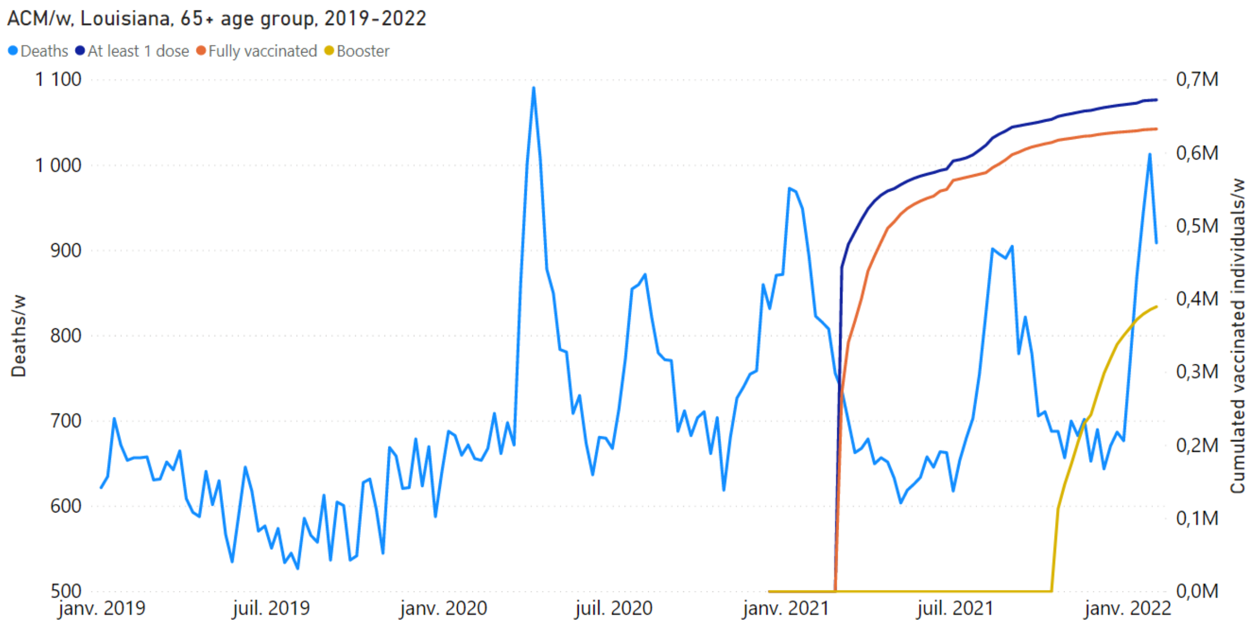
D


ACM/w, Florida, 25-64 age group, 2019-2022
Deaths At least 1 dose Fully vaccinated Booster
2 400
2 200
2 000
1 800
1 600
1 400
1 200
1 000
800
Deaths/w
10M
8M
6M
4M
2M
0M
Cumulated vaccinated individuals/w
janv. 2019
juil. 2019
janv. 2020
juil. 2020
janv. 2021
juil. 2021
janv. 2022

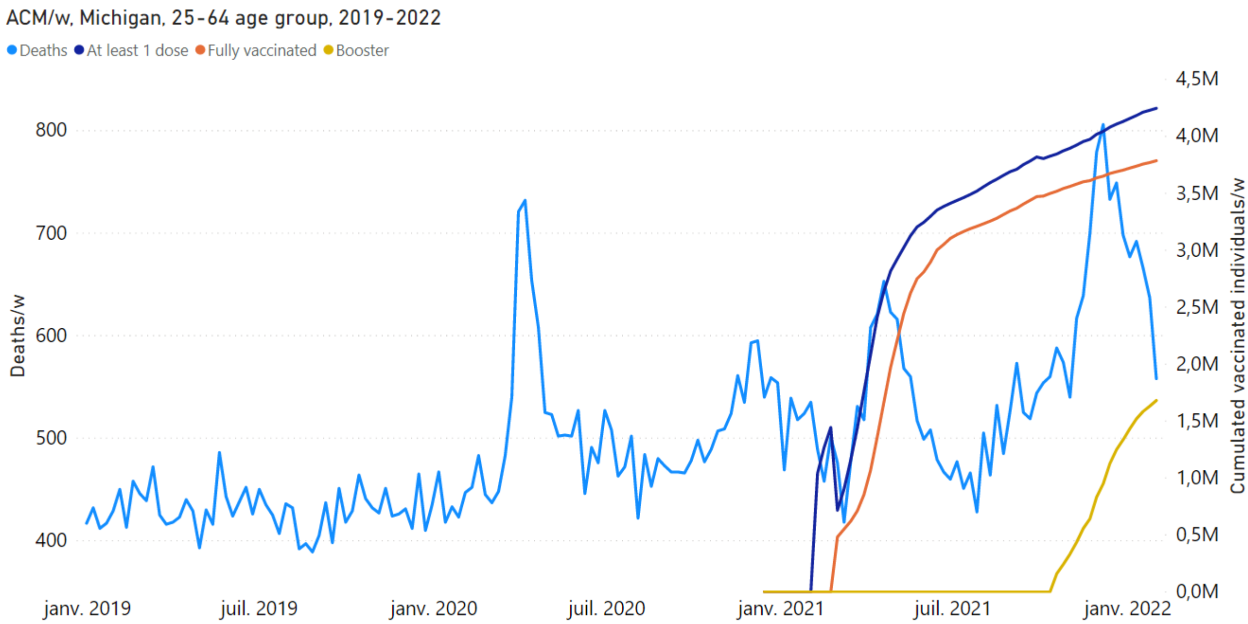
E


ACM/w, Louisiana, 25-64 age group, 2019-2022
Deaths
At least 1 dose
Fully vaccinated
Booster
Deaths/w
Cumulated vaccinated individuals/w
500
450
400
350
300
250
200
2,0M
1,5M
1,0M
0,5M
0,0M
janv. 2019
juil. 2019
janv. 2020
juil. 2020
janv. 2021
juil. 2021
janv. 2022

F


ACM/w, Louisiana, 65+ age group, 2019-2022
Deaths
At least 1 dose
Fully vaccinated
Booster
Deaths/w
Cumulated vaccinated individuals/w
1 100
1 000
900
800
700
600
500
0,7M
0,6M
0,5M
0,4M
0,3M
0,2M
0,1M
0,0M
janv. 2019
juil. 2019
janv. 2020
juil. 2020
janv. 2021
juil. 2021
janv. 2022

G



H

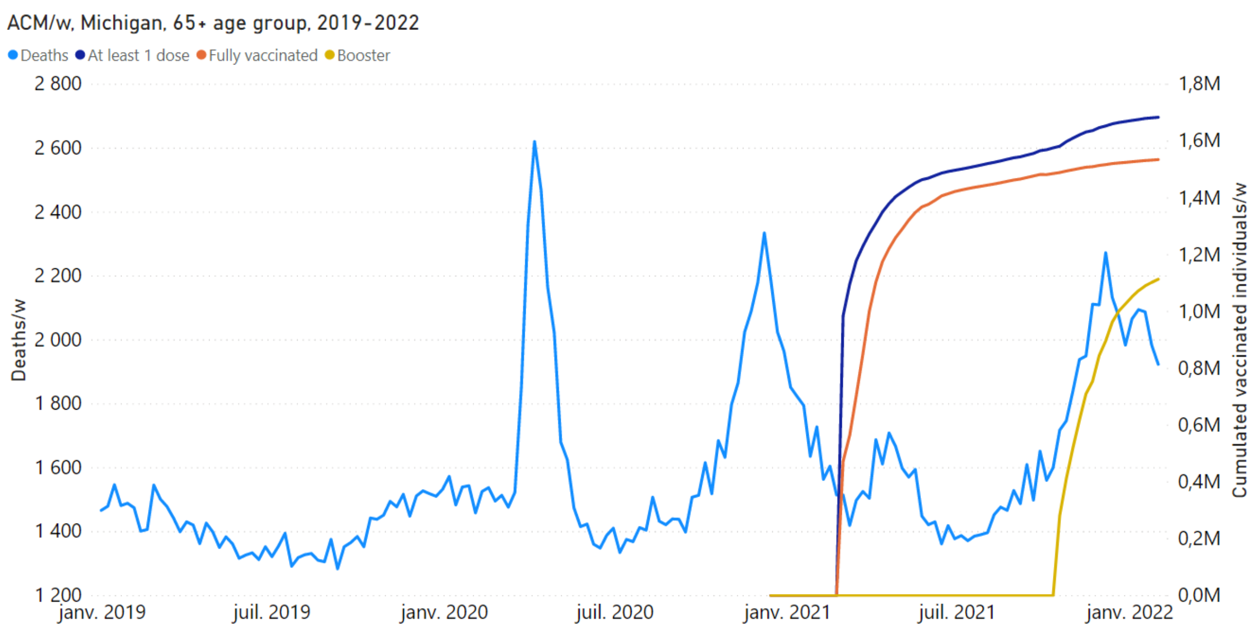


Figure 11 illustrates the late-summer-2021 peak in ACM/w for the states of Alabama, Mississippi, Georgia, Florida and Louisiana; and the unique spring-2021 (April-centered) peak in ACM/w occurring for Michigan.

Here, the “modest but significant stepwise increase in 1st-dose vaccine delivery, which is synchronous with the late-summer-2021 peak in ACM, visible for all ages and for the

25-44 and 45-64 years age groups (Figure 10A, C, D)" discussed above for the whole USA is now examined through the ACM/w and cumulative vaccine dose delivery by week data for the states of Alabama, Mississippi, Georgia, Florida and Louisiana, where the feature is prominent (Figure 11A, B, C, D, E, F), in the 25-64 years age group in particular. These five states are examples of states in which the late-summer-2021 peak is the most intense feature (largest peak) in the ACM/w data. In each case, the synchronous stepwise increase in cumulative vaccine dose delivery is evident.

This association between late-summer-2021 peak and stepwise increase in vaccine dose delivery is present throughout all the states: Where this is a most prominent late-summer-2021 peak there is an evident synchronous stepwise increase in vaccine dose delivery, and *vice versa*. The case of the state of Michigan shows a counter example: There is no late-summer-2021 peak and there is no stepwise increase in vaccination (Figure 11G, H).

However, the case of Michigan is shown for an additional reason: Michigan is the only state that has a spring-2021 (April-centered) peak in ACM/w (Figure 11G, H). This is arguably the most remarkable feature in all of the ACM data for the USA, since it occurs only in one state and does not correspond to a local intense summer heatwave phenomenon.

Michigan's said spring-2021 peak in ACM/w occurs synchronously with Michigan's fastest increase in vaccine dose delivery for 18-64 year olds (Figure 11G). It occurs when the vaccination campaign was "turned on" for this age group. This is also the time (April-2021) when, for this age group, for the whole USA, vaccine delivery was at its highest, and all reported vaccine adverse effects, including death, peaked (Hickey and Rancourt, 2022; their Figure S2). The Janssen-shot deliveries (shots administered), in particular, peaked strongly in approximately April-2021 (whole USA) (Hickey and Rancourt, 2022; their Figure S1), and were CDC-recommended to be "paused", and then re-authorized at approximately that time, also (FDA, 2021, 2022).

For Michigan, therefore, one is tempted to directly assign the unique spring-2021 peak in mortality as directly caused by the vaccine injections. The vaccine fatality toxicity per dose would need to be approximately 10 times greater than the known value for non-immunocompromised subjects (Hickey and Rancourt, 2022; their Table 1). However, if immunocompromised young adults (stressed and mentally disabled, and such, see below) were captured by the vaccination campaign, then the causal link is entirely possible.

Coming back to the big picture: The massive vaccination campaign in the USA did not reduce all-cause mortality to a pre-covid-period level, overall or in any of the age groups; nor does it appear to have substantially increased ACM during the vaccination campaign, compared to the pre-vaccination period of the covid period (Figure 10).

In the next section, we use the method described above (in section 3.2.2) to quantitatively assess whether the vaccination campaign measurably affected integrated ACM.

3.3.3. Quantifying excess mortality of the pre-vaccination and vaccination periods of the covid period, by age group

We adapt our method described in section 3.2.2 and use the ACM/w data of Figure 5 to quantify the excess mortality of the vaccination period "to date", compared to the excess mortality of the pre-vaccination period of the covid period, as follows.

The idea is to test whether there is a significant systematic increase in mortality, by state and by age group, occurring after the large increase in vaccination injections, compared to the (equal duration) part of the covid period prior to the surge in vaccination delivery, and compared to a pre-covid period of same duration occurring immediately prior to the 11 March 2020 start of the covid period.

For a given age group, we add all the weekly deaths together, for the weeks of 22 February 2021 (week-8 of 2021, inflection point of the vaccination period) through to the latest useable week (week-5 of 2022, beginning of February 2022). This is a total for 50 weeks (the vaccination period “to date”). In analogy with our previously introduced notation (above in section 3.2.2), we call this total “w50c”. Then we perform a similar total for the 1st-prior 50-week period, immediately preceding the vaccination period, for the 50 weeks up to and including week-7 of 2021. We call this total “w50c-1”. These two 50-week periods of the covid period, divide the covid period into equal-duration pre-vaccination (w50c-1) and vaccination (w50c) periods, which can be visualized with the help of Figure 10A and Figure 12 (below). And we do the same for the 2nd-prior 50-week period, and we call this total “w50c-2”. We continue moving back in time, to the end of the useable data in 50-week periods: w50c-3, etc.

Figure 12 shows the graph of “w50c-x” versus time, together with the ACM/w for the USA where each 50-week period is distinguished using a different color.

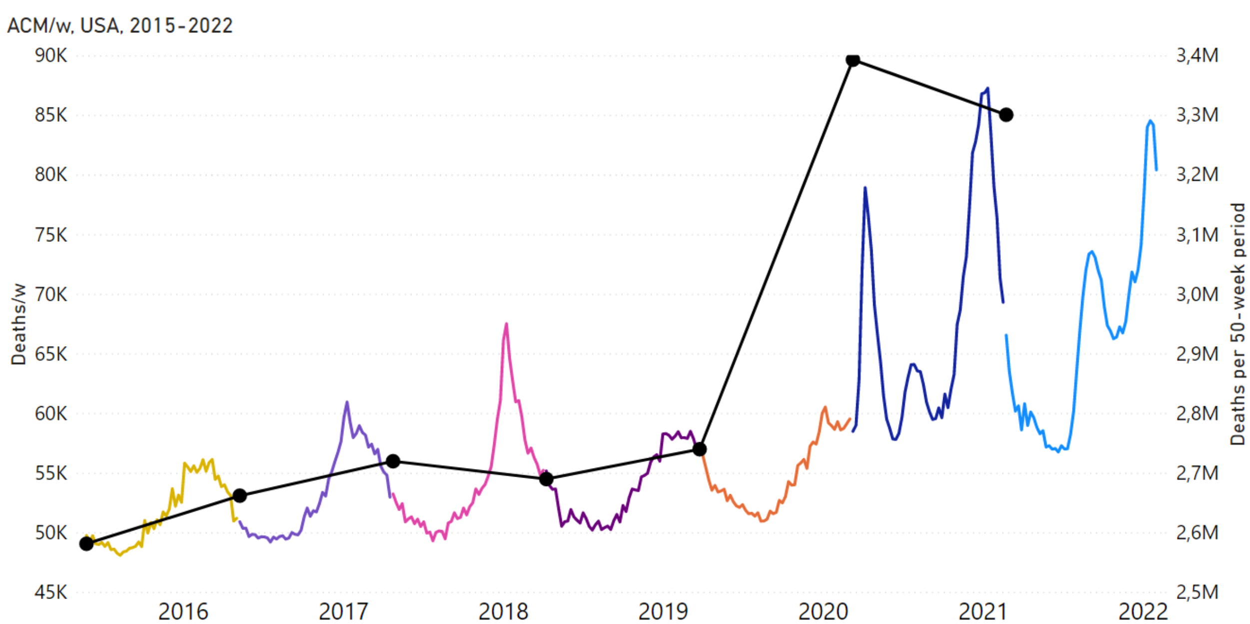


Figure 12. All-cause mortality by week (colors) and by 50-week period (black) in the USA from 2015 to 2022. Data are displayed from week-21 of 2015 to week-5 of 2022. The different colors indicate the successive 50-week periods. The light-blue color corresponds to the vaccination period of the covid period. The dark-blue color corresponds to the pre-vaccination period of the covid period. All the other colors are in the pre-covid period. The black dots show

the integrated ACM on these 50-week periods. Data were retrieved from CDC (CDC, 2022b), as described in Table 1.

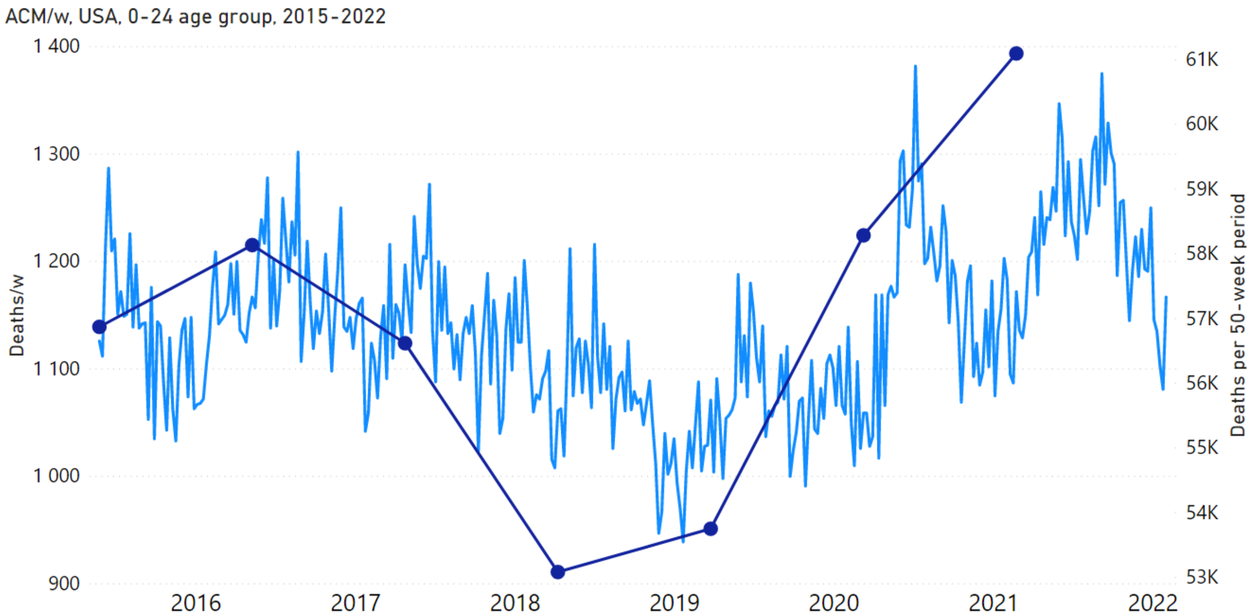
Equivalents to Figure 12 (without the color-code) for each of the states of the USA can be found in Appendix A.

Contrary to what would be expected if we assumed that the injections themselves induced a large (dominant) measurable positive or negative change in ACM, over a 50-week integration period the integrated ACM in the vaccination period of the covid period is comparable to and lower than in the pre-vaccination period of the covid period, for the USA as a whole (Figure 12). Indeed, there is a much greater and discontinuous change in ACM in going between the pre-covid period and the covid period than in going between the pre-vaccination period of the covid period and the vaccination period of the covid period.

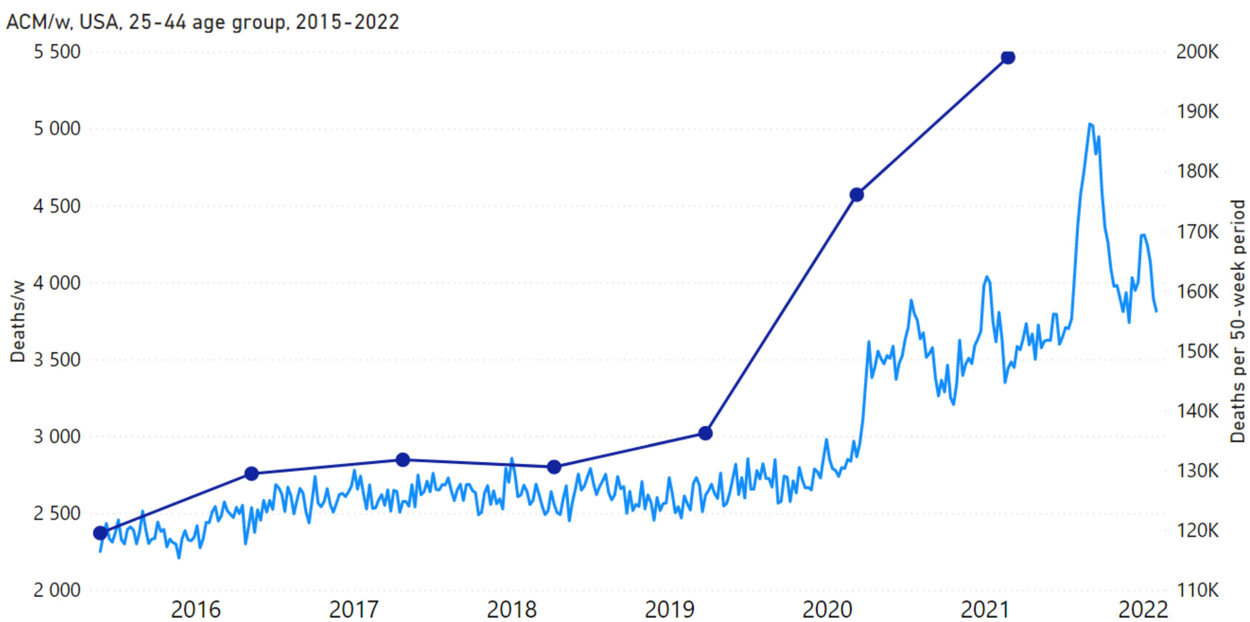
The mortality data (Figure 12) can be resolved by age group, which is shown, as follows, in Figure 13.

Figure 13. All-cause mortality by week (light-blue) and by 50-week period (dark-blue) in the USA from 2015 to 2022, for each of the age groups. Data are displayed from week-21 of 2015 to week-5 of 2022. Panels below: (A) for the 0-24 years age group; (B) for the 25-44 years age group; (C) for the 45-64 years age group; (D) for the 65-74 years age group; (E) for the 75-84 years age group; (F) for the 85+ years age group. Data were retrieved from CDC (CDC, 2022b), as described in Table 1.

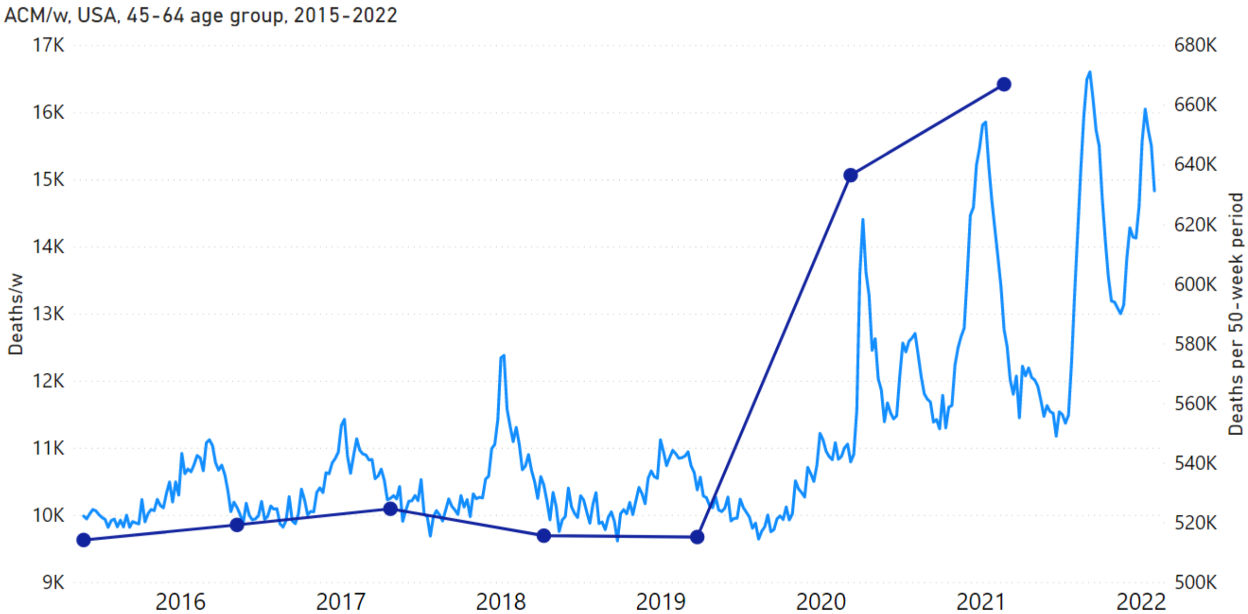
A


ACM/w, USA, 0-24 age group, 2015-2022
1 400
1 300
1 200
1 100
1 000
900
Deaths/w
61K
60K
59K
58K
57K
56K
55K
54K
53K
Deaths per 50-week period
2016
2017
2018
2019
2020
2021
2022

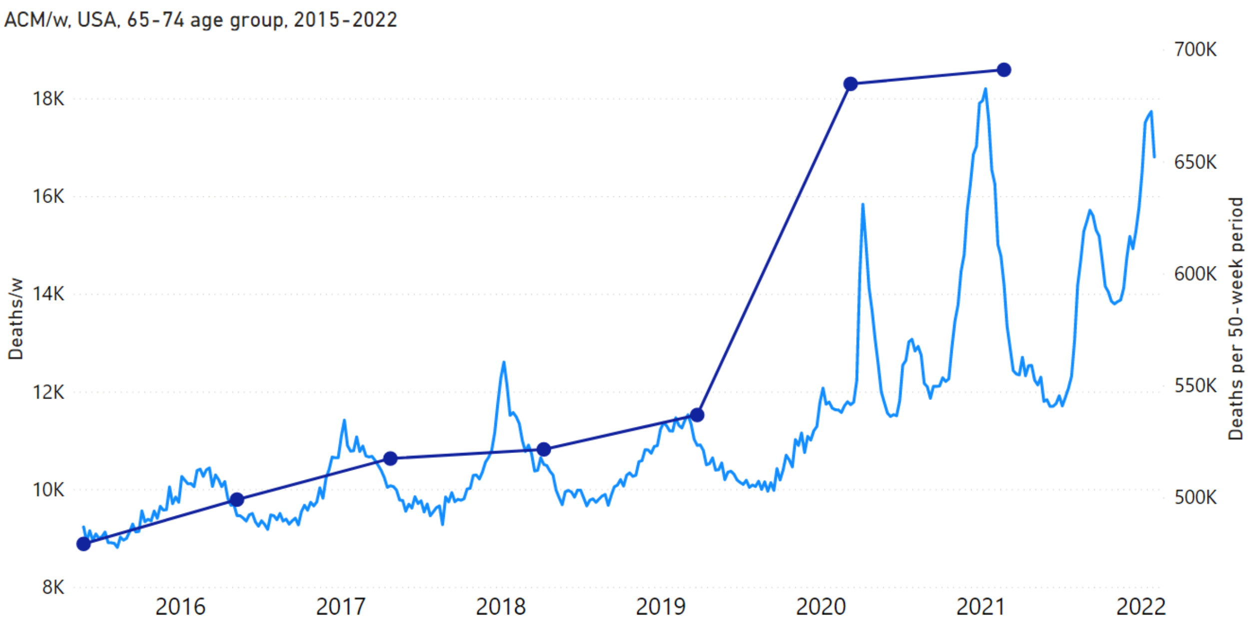
B


ACM/w, USA, 25-44 age group, 2015-2022
5 500
5 000
4 500
4 000
3 500
3 000
2 500
2 000
Deaths/w
200K
190K
180K
170K
160K
150K
140K
130K
120K
110K
Deaths per 50-week period
2016
2017
2018
2019
2020
2021
2022

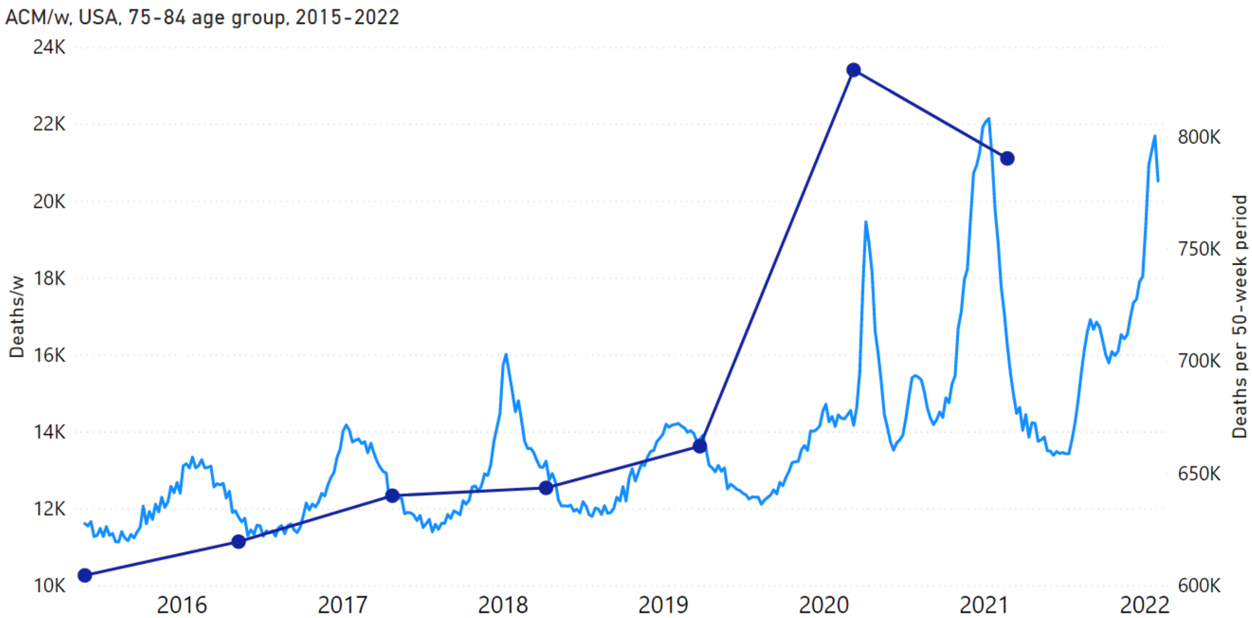
C


ACM/w, USA, 45-64 age group, 2015-2022
17K
16K
15K
14K
13K
12K
11K
10K
9K
Deaths/w
2016
2017
2018
2019
2020
2021
2022
680K
660K
640K
620K
600K
580K
560K
540K
520K
500K
Deaths per 50-week period

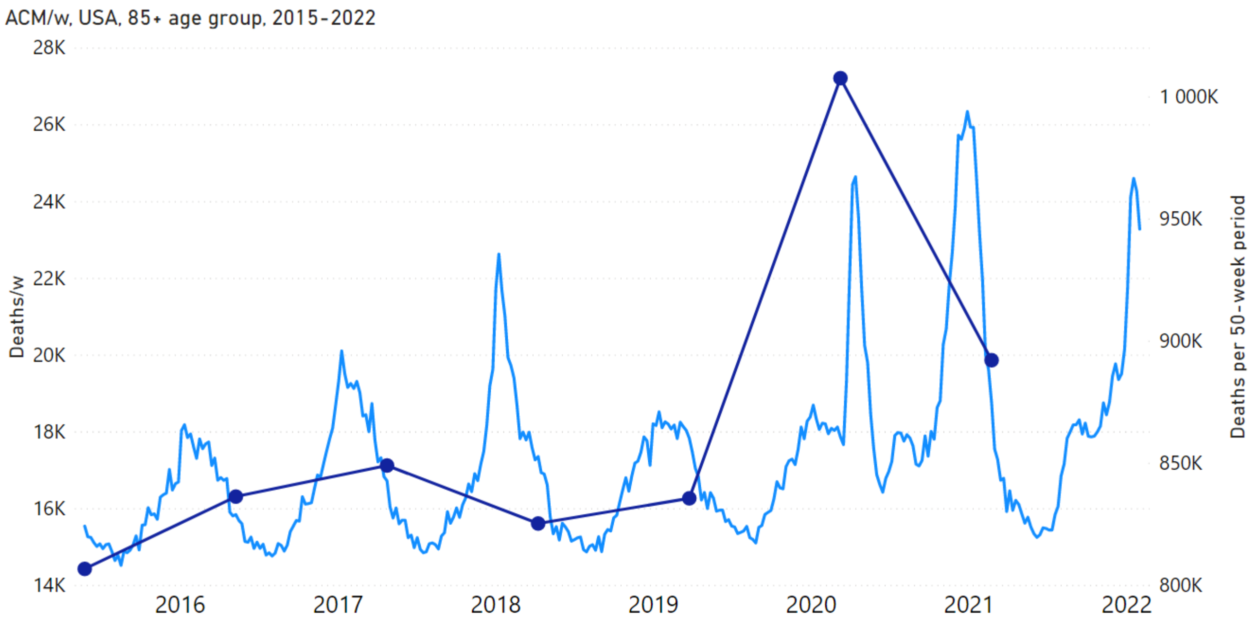
D


ACM/w, USA, 65-74 age group, 2015-2022
18K
16K
14K
12K
10K
8K
Deaths/w
2016
2017
2018
2019
2020
2021
2022
700K
650K
600K
550K
500K
Deaths per 50-week period

E



F



The ACM by 50-week period resolved by age group shows that integrated ACM is higher in the vaccination period of the covid period than in the pre-vaccination period of the covid period for all the younger age groups, under 75 years old (Figure 13A, B, C, D).

The integrated mortality by consecutive 50-week periods is shown for all the age groups together in Figure 14, by normalizing all the 50-week periods by the first 50-week period for each age group.

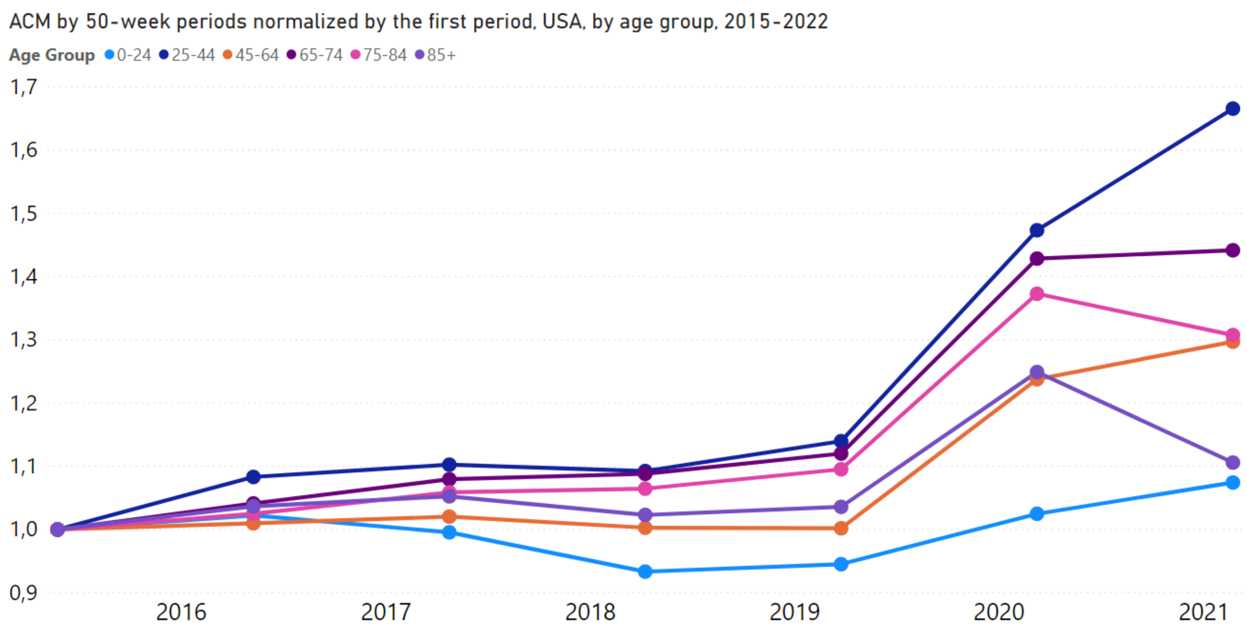


Figure 14. All-cause mortality by 50-week period normalized by the first 50-week period in the USA, from 2015 to 2022, for each of the age groups. Data are displayed from week-21 of 2015 to week-8 of 2021 (beginning of the vaccination period). ACM data were retrieved from CDC (CDC, 2022b), as described in Table 1.

The only age groups for which ACM in the vaccination period of the covid period is lower than ACM in the pre-vaccination period of the covid period are the 75-84 and 85+ age groups. All the other age groups show otherwise (Figure 14).

In order to quantify and directly compare the pre-vaccination period and the vaccination period within the covid period, we define the following quantities:

pVax-pCVD/pCVD = (w50c-1 - w50c-2) / w50c-2, expressed as a percentage, (9)

and

Vax-pCVD/pCVD = (w50c - w50c-2) / w50c-2, expressed as a percentage, (10)

Where w50c is the integrated ACM of the vaccination period of the covid period (50 weeks), w50c-1 the integrated ACM of the pre-vaccination period of the covid period (50 weeks) and w50c-2 the integrated ACM of the first pre-covid period of 50 weeks (immediately preceding the covid period).

Table 4 contains the calculated vaccination-period excess mortality (Vax-pCVD) and pre-vaccination-period excess mortality (pVax-pCVD) of the covid period, for each age group for the USA, and for the entire USA ("Total"), and the relative changes also, using each equation described above (Equations 9 and 10), as percentages of the pre-covid-period reference values (w50c-2).

Age Group	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
0-24	61 336	58 285	53 751	4 534	7 585	8,44 %	14,11 %
25-44	199 698	176 183	136 281	39 902	63 417	29,28 %	46,53 %
45-64	668 308	636 579	515 280	121 299	153 028	23,54 %	29,70 %
65-74	692 322	684 913	537 036	147 877	155 286	27,54 %	28,92 %
75-84	791 625	830 068	662 236	167 832	129 389	25,34 %	19,54 %
85+	893 194	1 007 727	835 708	172 019	57 486	20,58 %	6,88 %
Total	**3 306 483**	**3 393 755**	**2 740 292**	**653 463**	**566 191**	**23,85 %**	**20,66 %**

Table 4. Estimated excess mortality of the pre-vaccination and vaccination periods of the covid period in the USA, by age group. w50c is the total deaths during the vaccination period of the covid period (from week-8 of 2021 to week-5 of 2022, included). w50c-1 is the total deaths during the pre-vaccination period of the covid period (from week-11 of 2020 to week-7 of 2021, included). w50c-2 is the total deaths during the pre-covid period (from week-13 of 2019 to week-10 of 2020, included). pVax-pCVD and Vax-pCVD correspond to the excess mortality in the pre-vaccination period of the covid period and to the excess mortality in the vaccination period of the covid period, respectively. pVax-pCVD/pCVD and Vax-pCVD/pCVD correspond to the relative changes, as percentages of the pre-covid-period mortality, calculated from Equation 9 and Equation 10, respectively. ACM data were retrieved from CDC (CDC, 2022b), as described in Table 1.

Equivalents to Table 4 for each of the states of the USA can be found in Appendix A.

The numbers in Table 4 are represented graphically in bar charts, below, and are discussed below.

Figure 15 shows those quantities together with the relative excess mortality change in the covid period (xDc(100)1%, Equation 7) for each of the age groups for the whole USA.

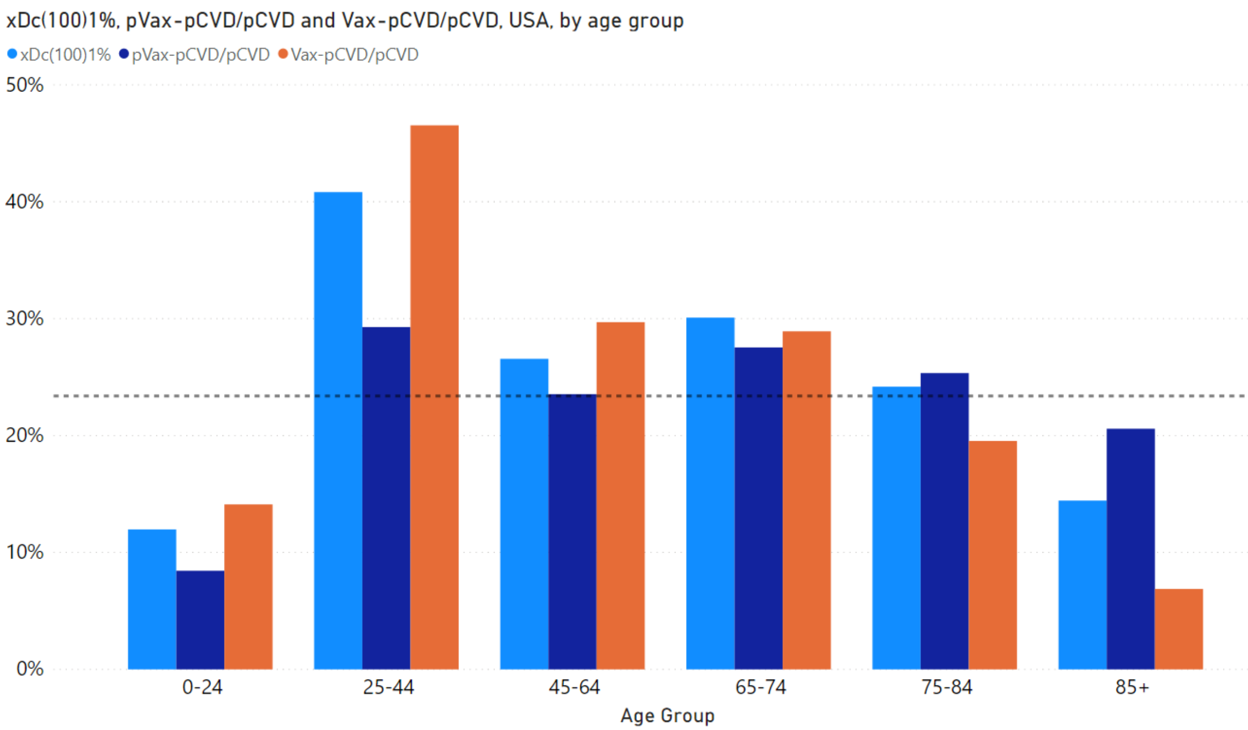


Figure 15. Excess mortality of the covid period (xDc(100)1%) (light-blue), of the pre-vaccination period of the covid period (pVax-pCVD/pCVD) (dark blue) and of the vaccination period of the covid period (Vax-pCVD/pCVD) (orange) in the USA, as percentages of the pre-covid-period mortality, by age group. The constant dashed line represents the value of xDc(100)1% for the whole USA. ACM data were retrieved from CDC (CDC, 2022b), as described in Table 1. xDc(100)1%, pVax-pCVD/pCVD and Vax-pCVD/pCVD are calculated from Equation 7, Equation 9 and Equation 10, respectively.

The excess mortality in the pre-vaccination period of the covid period is relatively lower than the excess mortality in the vaccination period of the covid period and lower than the excess mortality of the covid period for the younger age groups (0-24, 25-44, 45-64, 65-74) (Figure 15). The opposite is true for the older ages (75-84, 85+ years) (Figure 15). This qualitative difference can be interpreted as possibly associated to the

vaccination program, along the lines discussed above (Figure 10; Figure 11), in relation to the late-summer-2021 peak and the synchronous modest stepwise increase in cumulative vaccine dose delivery (administered). However, it is also possible that the said qualitative difference results instead (or concomitantly) as being due to the impacts of cumulative socio-economic pressures. Younger adults will have more resilience than older adults, such that the deadly toll of life-changing circumstances will take longer to materialize.

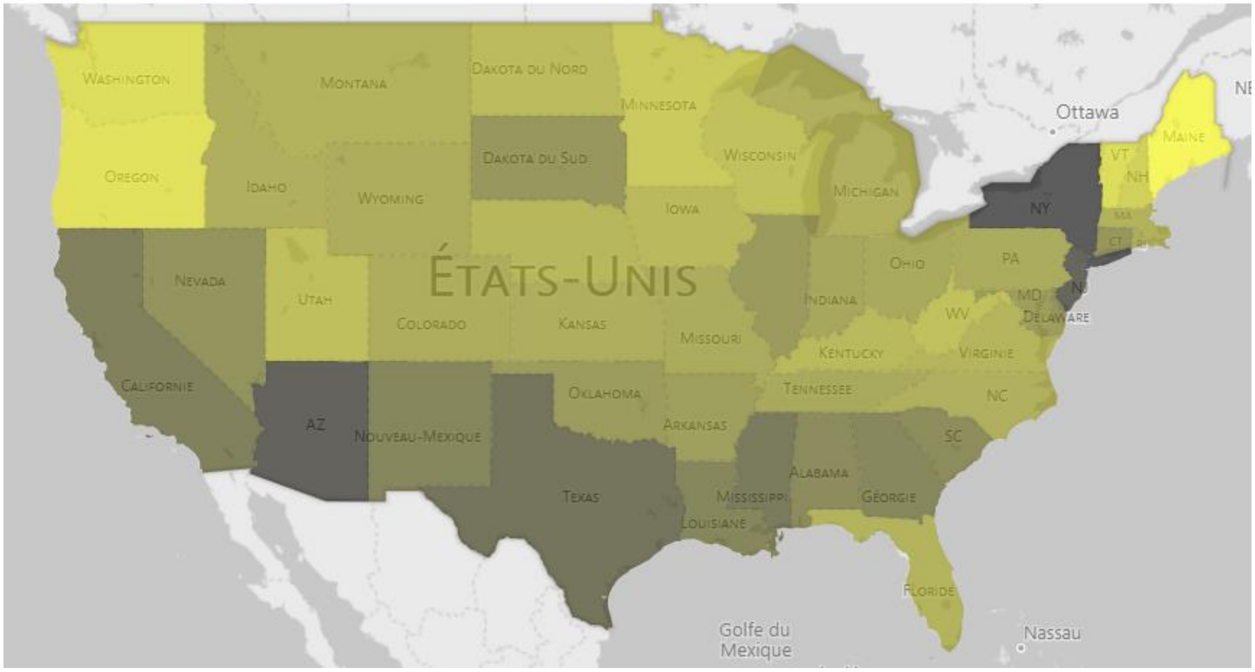
Next, we look at the excess mortality in the pre-vaccination period of the covid period and in the vaccination period of the covid period at the state level.

3.3.4. Excess mortality of the pre-vaccination and vaccination periods of the covid period, by state

Figure 16 shows USA maps of the covid-period pre-vaccination-period relative excess mortality (pVax-pCVD/pCVD) (Panel A) and of the covid-period vaccination-period relative excess mortality (Vax-pCVD/pCVD) (Panel B), as relative changes in percentages of the pre-covid-period mortality by state.

A

PVAX-PCVD/PCVD IN THE USA



B

VAX-PCVD/PCVD IN THE USA

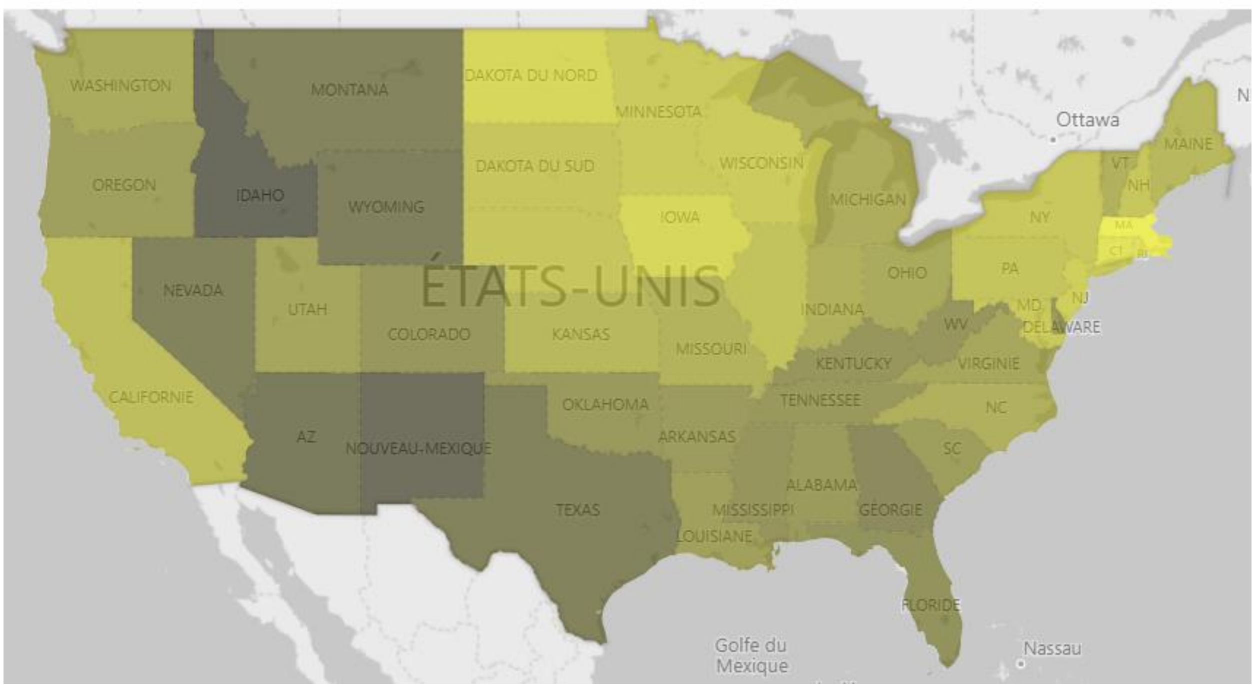


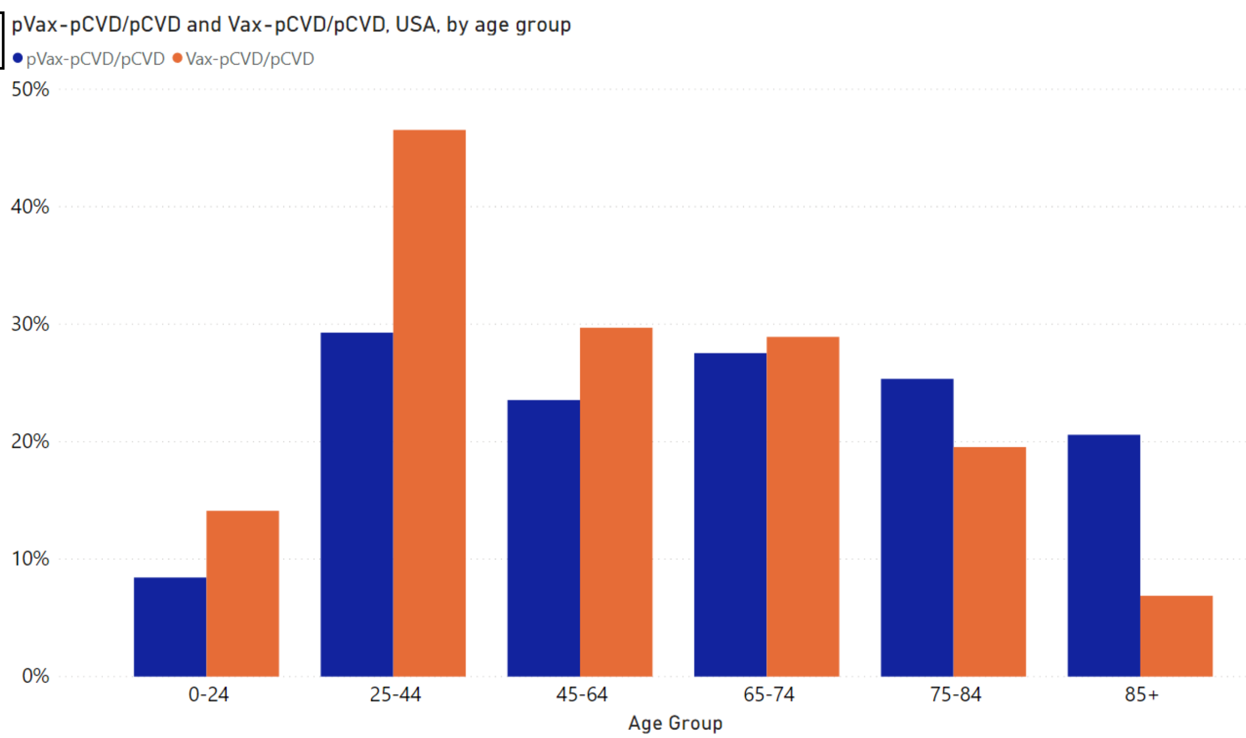
Figure 16. Maps of the excess mortality in the pre-vaccination period of the covid period (panel A) and in the vaccination period of the covid period (panel B) in the USA, as percentages of the pre-covid-period mortality. Alaska and Hawaii are excluded. The darker the color (black), the more intense is the relative change. ACM data were retrieved from CDC

(CDC, 2022b), as described in Table 1. pVax-pCVD/pCVD and Vax-pCVD/pCVD are calculated from Equation 9 and Equation 10, respectively.

Figure 16 shows a striking "positive-negative" effect in which many states that have relatively large relative mortality in the first half of the covid period (Panel A) have a relatively small relative mortality in the second half of the covid period (Panel B), and *vice versa*. This suggests a long-term (2 year) "dry tinder effect" in which vulnerable populations are decimated early or late during the 100-week covid period, but that once decimated cannot be re-decimated.

Figure 17 shows the covid-period pre-vaccination-period excess mortality (Equation 9) and the covid-period vaccination-period excess mortality (Equation 10) as percentages of the pre-covid-period mortality by age group, for the whole USA (Panel A), and for the ten most populous states (Panels B and C), ordered from the most populous to the less populous (US Census Bureau, 2022a): California, Texas, Florida, New York, Pennsylvania, Illinois, Ohio, Georgia, North Carolina and Michigan.

A



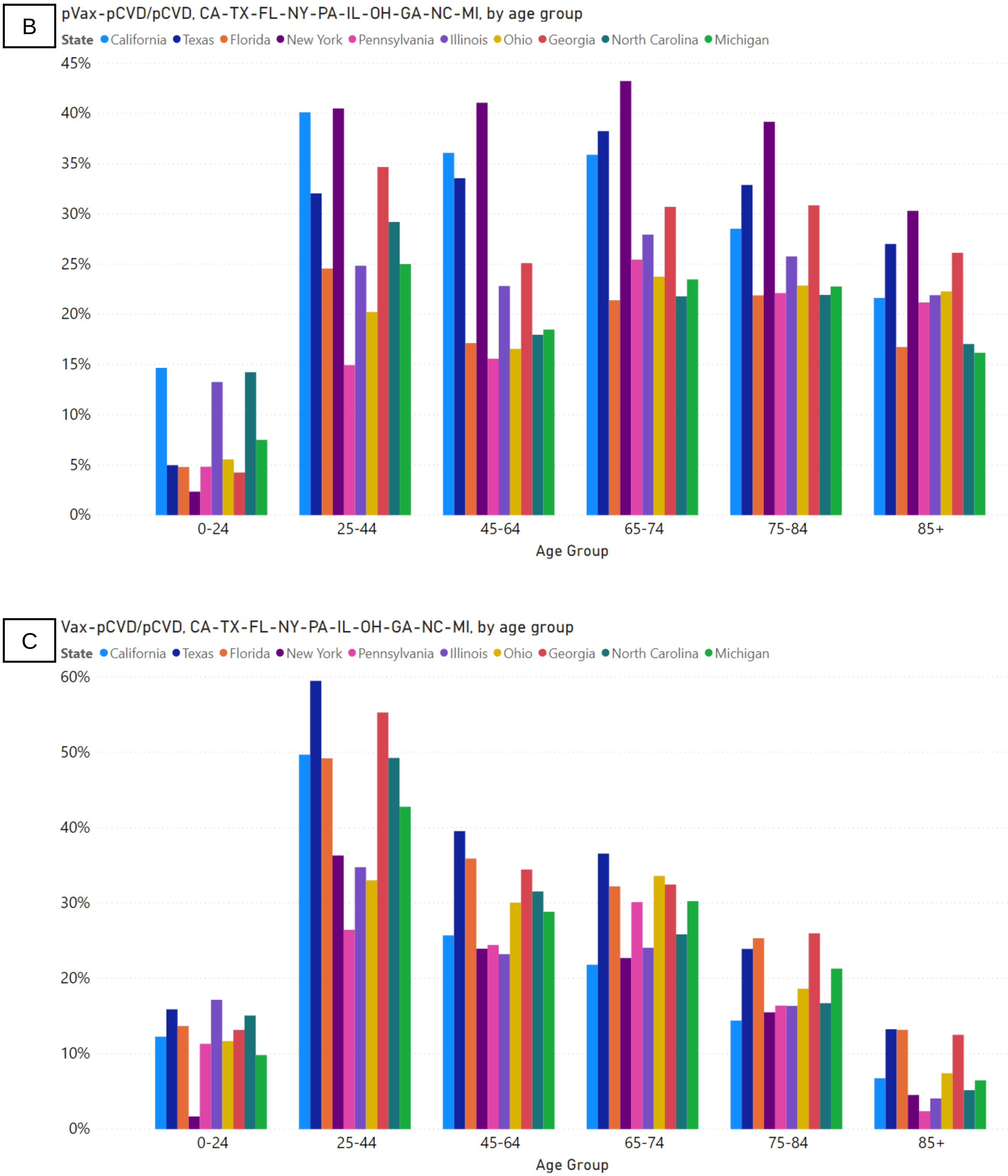


Figure 17. Excess mortality in the pre-vaccination period of the covid period (pVax-pCVD/pCVD) and in the vaccination period of the covid period (Vax-pCVD/pCVD) in the USA (Panel A) and in the ten most populous states of the USA (from left to right in each band: California, Texas, Florida, New York, Pennsylvania, Illinois, Ohio, Georgia, North

Carolina, Michigan) for the pre-vaccination period of the covid period (Panel B) and for the vaccination period of the covid period (Panel C), as percentages of the pre-covid-period mortality, by age group. ACM data were retrieved from CDC (CDC, 2022b), as described in Table 1. pVax-pCVD/pCVD and Vax-pCVD/pCVD are calculated from Equation 9 and Equation 10, respectively.

Figure 15 and Figure 17 strikingly illustrate a large systematic change in going between the pre-vaccination period of the covid period (first 50 weeks) and the vaccination period of the covid period (second 50 weeks): The age structure of relative excess mortality changes significantly, from being largely uniform with age (pre-vaccination) to being highly weighted towards young adults (vaccination).

Regarding the evident change in age structure of the relative mortality in going from the pre-vaccination period of the covid period into the vaccination period of the covid period (Figure 17), the same possible interpretations apply as discussed above for Figure 15: The said change in age structure can be interpreted as possibly associated to the vaccination program, along the lines discussed above (Figure 10; Figure 11), in relation to the late-summer-2021 peak and the synchronous modest stepwise increase in cumulative vaccine dose delivery (administered). However, it is also possible that the said change in age structure results instead (or concomitantly) as being due to the impacts of cumulative socio-economic pressures. Younger adults will have more resilience than older adults, such that the deadly toll of life-changing circumstances will take longer to materialize. Both of these hypotheses (resilience in youth and vaccine assault of vulnerable-group individuals), in turn, are consistent with the fact that the prevalence of serious mental illness is large and highly skewed towards young adults in the USA (NIMH, 2022).

In the next section, we explore the differential integrated mortality between the vaccination and pre-vaccination periods of the covid period at the state level.

3.3.5. Difference of vaccination and pre-vaccination mortality in the covid period, by age group and by state

For a given age group and state, we calculate the difference (Vax-pVax) between integrated mortality in the vaccination period of the covid period (w50c) and integrated mortality in the pre-vaccination period of the covid period (w50c-1):

Vax-pVax = w50c - w50c-1 (11)

This difference (Vax-pVax) normalized by the pre-covid-period integrated mortality (w50c-2) is:

Vax-pVax/pCVD = (w50c - w50c-1) / w50c-2, expressed as a percentage, (12)

Table 5 contains the calculated difference in mortality between the vaccination and pre-vaccination periods of the covid period (Vax-pVax), for each age group for the USA, and for the entire USA ("Total"), and the relative change also, as percentages of the pre-covid-period reference values (w50c-2).

Age Group	w50c	w50c-1	w50c-2	Vax-pVax	Vax-pVax/pCVD
0-24	61 336	58 285	53 751	3 051	5,68 %
25-44	199 698	176 183	136 281	23 515	17,25 %
45-64	668 308	636 579	515 280	31 729	6,16 %
65-74	692 322	684 913	537 036	7 409	1,38 %
75-84	791 625	830 068	662 236	-38 443	-5,81 %
85+	893 194	1 007 727	835 708	-114 533	-13,70 %
Total	**3 306 483**	**3 393 755**	**2 740 292**	**-87 272**	**-3,18 %**

Table 5. Difference of vaccination and pre-vaccination mortality in the covid period in the USA, by age group. w50c is the total deaths during the vaccination period of the covid period (from week-8 of 2021 to week-5 of 2022, included). w50c-1 is the total deaths during the pre-vaccination period of the covid period (from week-11 of 2020 to week-7 of 2021, included). w50c-2 is the total deaths during the pre-covid period (from week-13 of 2019 to week-10 of

2020, included). Vax-pVax corresponds to the difference between the vaccination-period mortality and the pre-vaccination-period mortality, calculated from Equation 11. Vax-pVax/pCVD corresponds to the relative change, as percentage of the pre-covid-period mortality, calculated from Equation 12. ACM data were retrieved from CDC (CDC, 2022b), as described in Table 1.

Equivalents to Table 5Table 4 for each of the states of the USA can be found in Appendix A.

In the covid period, vaccination-period ACM is greater than pre-vaccination-period ACM for younger people, and smaller for older people (Table 5). In terms of deaths predominantly caused by the vaccines, this would be opposite to the known exponential increase with age of vaccine-associated deaths (Hickey and Rancourt, 2022).

Figure 18 shows a USA map of the state-wise difference between vaccination and pre-vaccination mortality (Vax-pVax), as relative changes in percentage of the pre-covid-period mortality (Vax-pVax/pCVD).

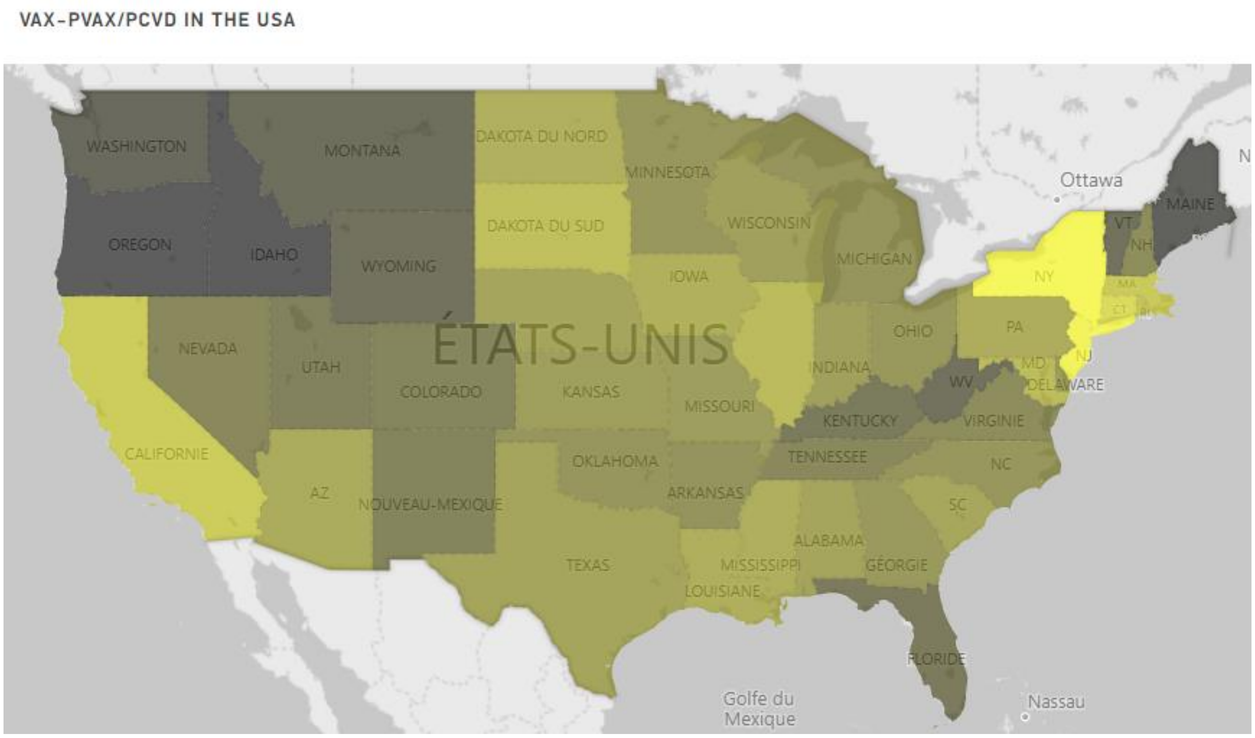


Figure 18. Map of the difference of vaccination and pre-vaccination mortality in the covid period in the USA, as percentages of the pre-covid-period mortality. Alaska and Hawaii are

excluded. The darker the color (black or yellow), the more intense is the relative change (positive or negative, respectively). ACM data were retrieved from CDC (CDC, 2022b), as described in Table 1. Vax-pVax/pCVD is calculated from Equation 12.

Figure 18 is a geographical representation of where (by state) the differences between the mortality per pre-covid mortality (pCVD) of the first half of the covid period (pVax; pre-vaccination period) and the second half of the covid period (Vax; vaccination period) are largest, both negative (pVax > Vax; darkest yellow) and positive (Vax > pVax; darkest grey). The known initial hot spots of New Jersey and New York are bright yellow, whereas the states with comparatively large late-covid-period mortality show up in dark grey: Maine, Oregon, Idaho, Washington, Florida…

In our view, it is not tenable to propose that the structure represented in Figure 18 arises from the national vaccination campaign as the dominant causal factor. There is no logical reason to propose, as the dominant excess-mortality-determining factor, that the vaccines saved lives in the states that have the largest initial (first 50 weeks of the covid period) mortality per capita or per pre-covid mortality and/or caused massive mortality per capita or per pre-covid mortality in the states that had relatively small initial covid-period mortality per capita. However, the map (Figure 18) does suggest a “dry tinder effect” for vulnerable populations, over the course of approximately two years under covid-period conditions, as discussed above for Figure 16.

Figure 19 shows the Vax-pVax/pCVD (Equation 12) values from Table 5 by age group, for the whole USA (Panel A), and for the ten most populous states (Panel B), ordered from the most populous to the less populous (US Census Bureau, 2022a): California, Texas, Florida, New York, Pennsylvania, Illinois, Ohio, Georgia, North Carolina and Michigan. The horizontal dashed line represents the value for the whole USA (all ages and all states).

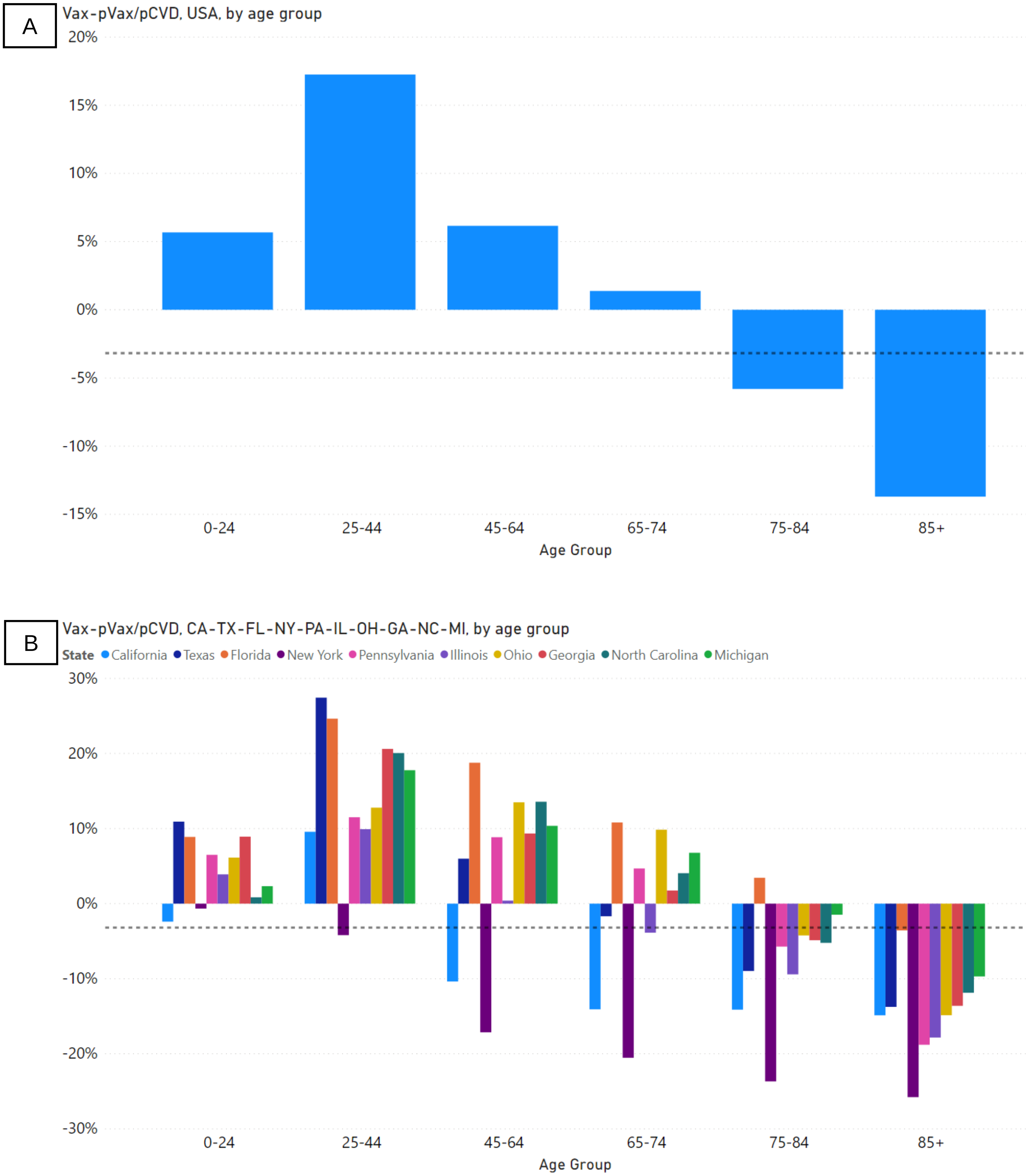


Figure 19. Difference of vaccination and pre-vaccination mortality in the covid period in the USA (panel A) and in the ten most populous states of the USA (from left to right in each band: California, Texas, Florida, New York, Pennsylvania, Illinois, Ohio, Georgia, North Carolina, Michigan) (panel B), as percentages of the pre-covid-period mortality, by age group. The constant dashed line represents the value for the whole USA. ACM data were

retrieved from CDC (CDC, 2022b), as described in Table 1. Vax-pVax/pCVD is calculated from Equation 12.

Figure 19 is another way (by difference) to illustrate the dramatic change in age structure of relative (i.e., age-group specific) excess mortality from being largely uniform with age (pre-vaccination) to being highly weighted towards young adults (vaccination), which is shown in Figure 17.

Figure 19 shows that more young adults died (relative to their population or to their pre-covid death rate) in the second half (Vax) of the covid period relative to the first half (pVax) of the covid period, for most states and for the whole USA. This is consistent with a long-term (2-year) “dry tinder effect” for elderly populations, and greater resilience against the assault of the covid-period conditions for younger populations, such as to take longer for mortality to be experienced in younger residents. It is also consistent with the hypothesis that immunocompromised young adults were captured by the vaccination campaign, including the so-called “vaccine equity” programs, which would also explain the large late-summer-2021 ACM peak for young adults discussed above. Both of these hypotheses, in turn, are consistent with the fact that the prevalence of serious mental illness is large and highly skewed towards young adults in the USA (NIMH, 2022).

Therefore, from all of the above, it does not appear that the USA vaccination campaign has had a dominant impact, positive or negative, on integrated all-cause mortality, although it may have participated or predominantly caused the change in age structure of mortality risk, and may have contributed to maintaining a large covid-period ACM. The changes in mortality per pre-covid mortality, which occur between the first (pVax) and second (Vax) halves of the covid period may be due to temporal changes in both quantity (“dry tinder effect”) and quality (age, resilience) of the vulnerable populations during a sustained covid-period assault on living conditions, and may have been significantly modulated by vaccine-campaign capture of immunocompromised young adults from vulnerable groups. In order to explore these hypotheses, regarding

vulnerable groups, we next quantify excess mortality per capita for the entire 100-week covid period and examine its correlations with various socio-economic factors, in the following section.

3.4. Associations of excess mortality of the covid period with socio-geo-economic variables

In our previous article (Rancourt, Baudin and Mercier, 2021b), we described associations of integrated excess (with respect to an extrapolated summer baseline mortality) all-cause mortality per capita in anomalous features (cvp1, smp1, cvp2, smp2) of all-cause mortality by time in the covid period with socio-geo-economic and climatic parameters:

> "[…] we have shown is that, in the COVID-era, during summer-2020 (smp1), fall-winter-2020-2021 (cvp2) and summer-2021 (smp2), combined factors including poverty, obesity and hot climate became deadly associations for excess (above-SB) deaths, beyond the deaths that would have occurred from the pre-COVID-era background of preexisting risk factors."

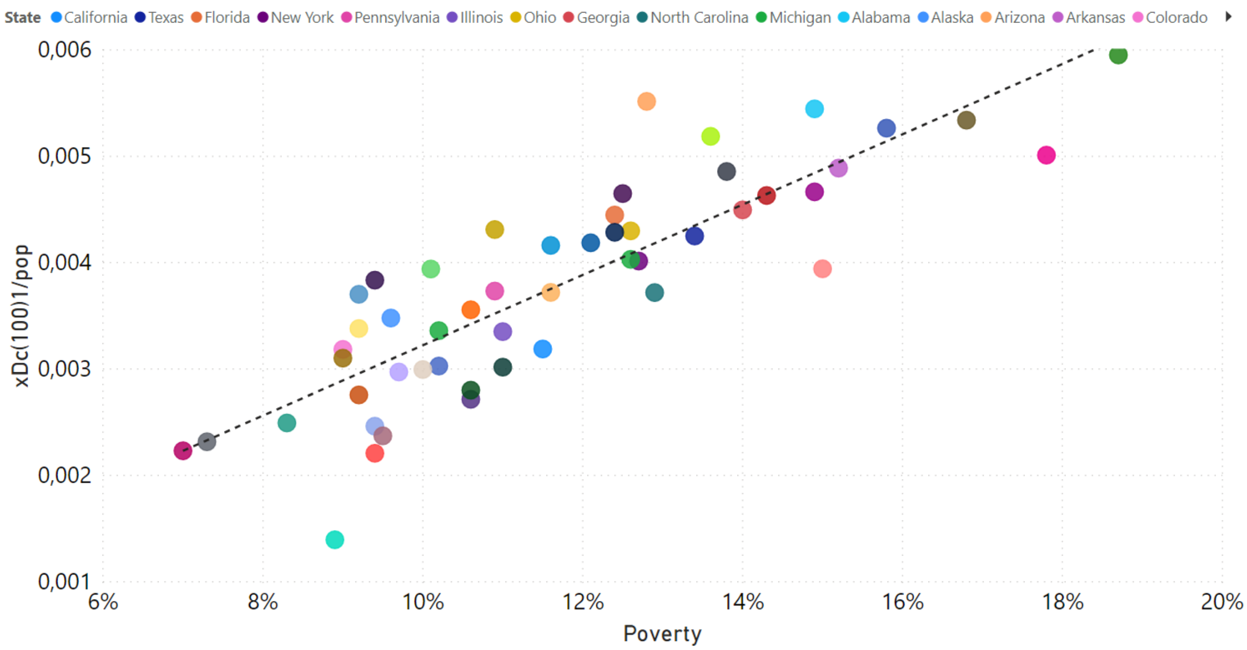
Therefore, here again we examine associations with such factors.

The following factors normalized by state population are tested against the quantified excess mortality of the covid period (xDc(100)1) normalized by the state population:

- Poverty
- Median Household Income (MHI)
- Obesity
- Population aged 65 and over (and 75+, and 85+)
- Supplemental Security Income (SSI)
- Social Security Disability Insurance (SSDI)
- Disability

Figure 20 shows the scatter plot for poverty (on two different scales, A and B), defined as the estimated percentage of the population of people of all ages living in poverty (US Census Bureau, 2022b). The Y-axis is the fraction xDc(100)1/pop, the 100-week covid-period excess mortality by population, which is the “100-week covid-period fatality ratio” for the USA population.

A



B

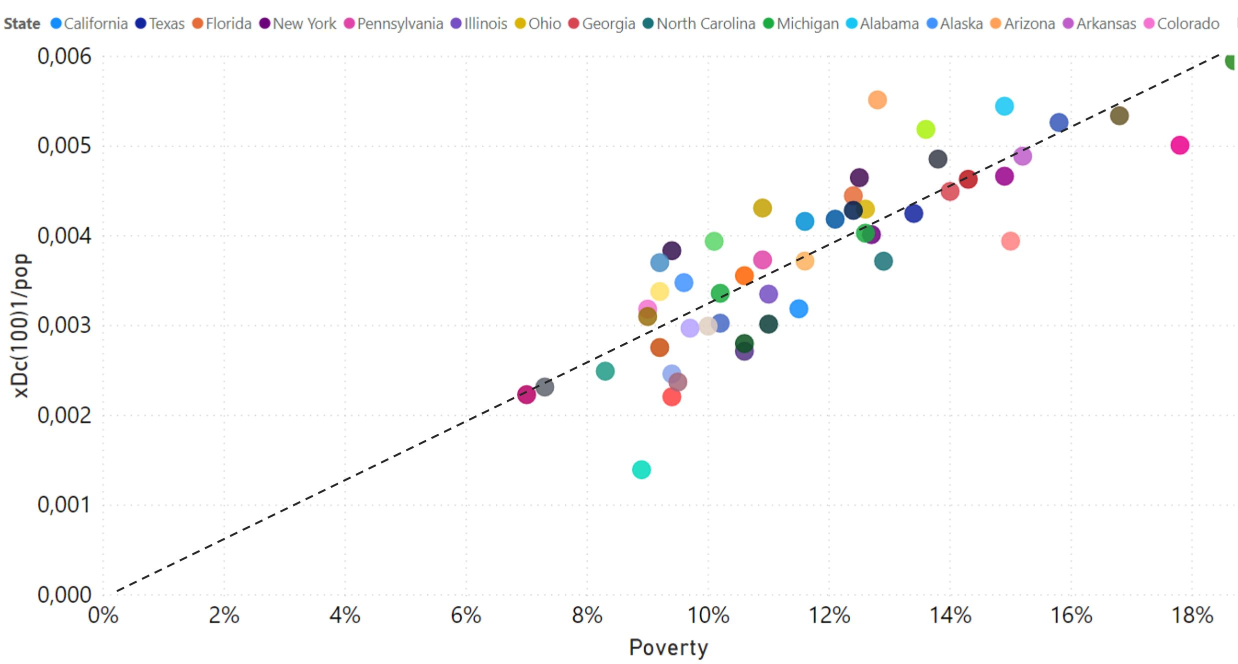


Figure 20. Excess mortality of the covid period normalized by population versus poverty in the USA. The axes are optimized for the dataset (Panel A) and for the intercept between trend line and X-axis (Panel B). Each point is for one state of the USA. The parameters of the least squares fitted linear trend line are given in Table 6. The color-code of the 51 states is shown in section 2. Data were retrieved as described in Table 1. xDc(100)1 is calculated from Equation 5.

Figure 20 is a striking result. Such a result is rarely so clear in epidemiological studies. The Pearson correlation coefficient is r = +0.86 (Table 6). Beyond this "very strong" correlation, we note that the least squares fitted straight line passes virtually through the origin (Table 6), implying that the integrated excess mortality per capita per state for the whole 100-week covid period (i.e., what we have termed the "100-week covid-period fatality ratio" for the USA population) is directly proportional to poverty of the state, not merely very strongly correlated to poverty. Such proportionality suggests a fundamental relationship, which is causal in nature; in which poverty captures or is an accurate proxy for the dominant factor or factors that determine mortality arising from all the conditions occuring during the covid period.

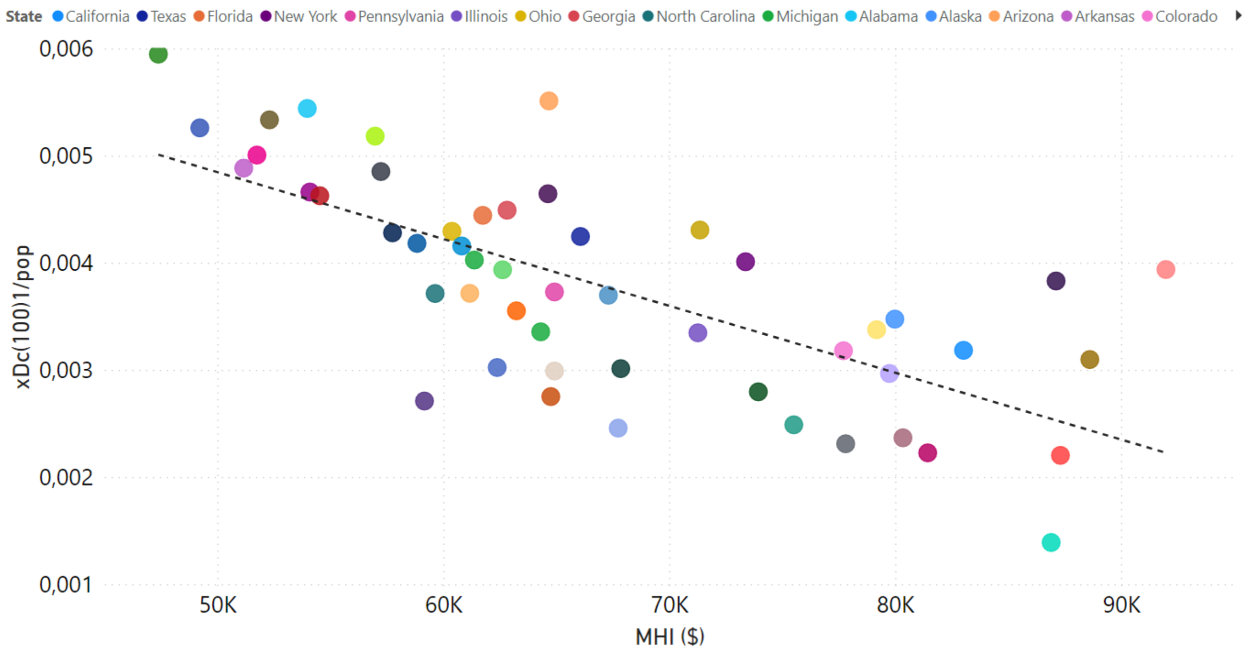
The said proportionality (Figure 20) means that a state with zero poverty would have experienced zero excess mortality in the 100-week covid period, and that doubling state-wise poverty (the fraction of state residents living in poverty) doubles excess mortality in the 100-week covid period, for example.

Furthermore, we note that it is unlikely that this strong epidemiological relationship with poverty arises from a viral respiratory disease. The classic development of a viral respiratory disease, leading to death, is one in which the infection fatality ratio is approximately exponential with age, with the main co-factors being comorbidity, not economic hardship itself, irrespective of age. There is no known viral respiratory disease in which the pathogen targets poverty, while being insensitive to age (see scatter plot versus age of the state population, Figure 23 below).

Figure 21 shows the scatter plot for median household income (MHI) (on two different scales, A and B), defined as the estimated median household income in US dollars (US

Census Bureau, 2022b). The Y-axis is the fraction xDc(100)1/pop, the 100-week covid-period excess mortality by population, which is the “100-week covid-period fatality ratio” for the USA population.

A



B

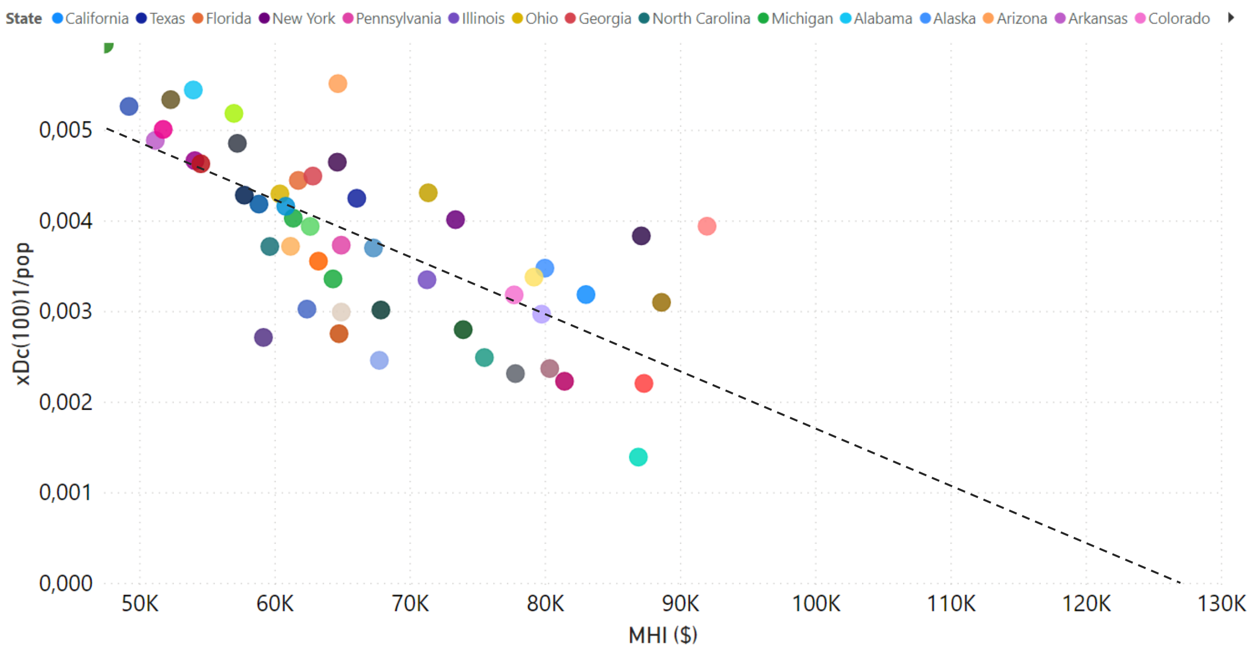


Figure 21. Excess mortality of the covid period normalized by population versus median household income (MHI) in the USA. The axes are optimized for the dataset (Panel A) and for the intercept between trend line and X-axis (Panel B). Each point is for one state of the USA.

The parameters of the least squares fitted linear trend line are given in Table 6. The color-code of the 51 states is shown in section 2. Data were retrieved as described in Table 1. xDc(100)1 is calculated from Equation 5.

Here, the Pearson correlation coefficient is r = −0.71 ("strong") (Table 6). The graph (Figure 21) suggests that a USA state with a MHI of approximately $130K or more would have zero excess mortality integrated over the 100-week covid period. Likewise, the states with smallest MHI attain a "100-week covid-period fatality ratio" of approximately 0.005, or 0.5%, which is very large, since this is over and above non-covid-induced mortality for such states.

Income (Figure 21) and poverty (Figure 20) are clearly determinative factors predicting excess 100-week covid-period mortality in a state of the USA, occuring since a pandemic was announced on 11 March 2020 by the WHO.

Figure 22 shows the scatter plot for obesity, defined as the prevalence of self-reported obesity among U.S. adults (CDC, 2021). The Y-axis is the fraction xDc(100)1/pop, the 100-week covid-period excess mortality by population, which is the "100-week covid-period fatality ratio" for the USA population.

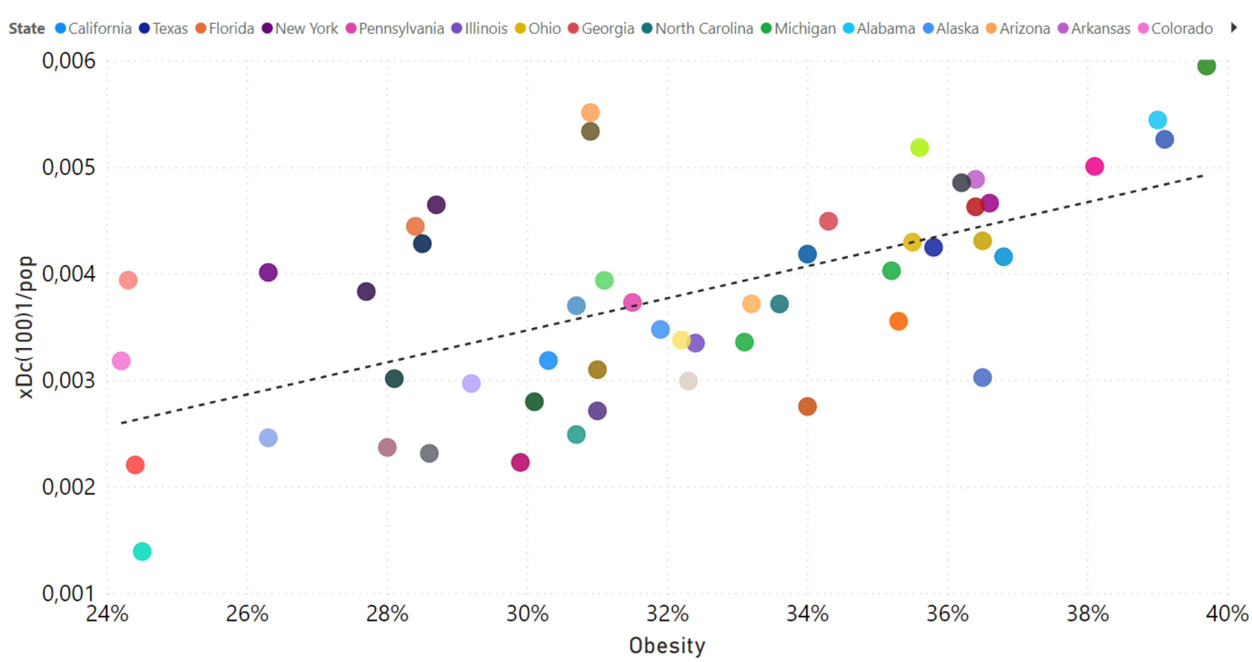


Figure 22. Excess mortality of the covid period normalized by population versus obesity in the USA. Each point is for one state of the USA. The parameters of the least squares fitted linear trend line are given in Table 6. The color-code of the 51 states is shown in section 2. Data were retrieved as described in Table 1. xDc(100)1 is calculated from Equation 5.

Here the positive correlation is “strong”, although less than for MHI, at r = +0.62 (Table 6). The least squares fitted straight line suggests that a USA state that would have an obesity rate of approximately 7% or less would have zero excess 100-week covid-period mortality. This implies that certain groups of obese residents do not contribute to 100-week covid-period excess mortality, presumably wealthy obese residents, for example.

Figure 23 shows the scatter plot for the proportion of the population aged 65 years old and over. The Y-axis is the fraction xDc(100)1/pop, the 100-week covid-period excess mortality by population, which is the “100-week covid-period fatality ratio” for the USA population.

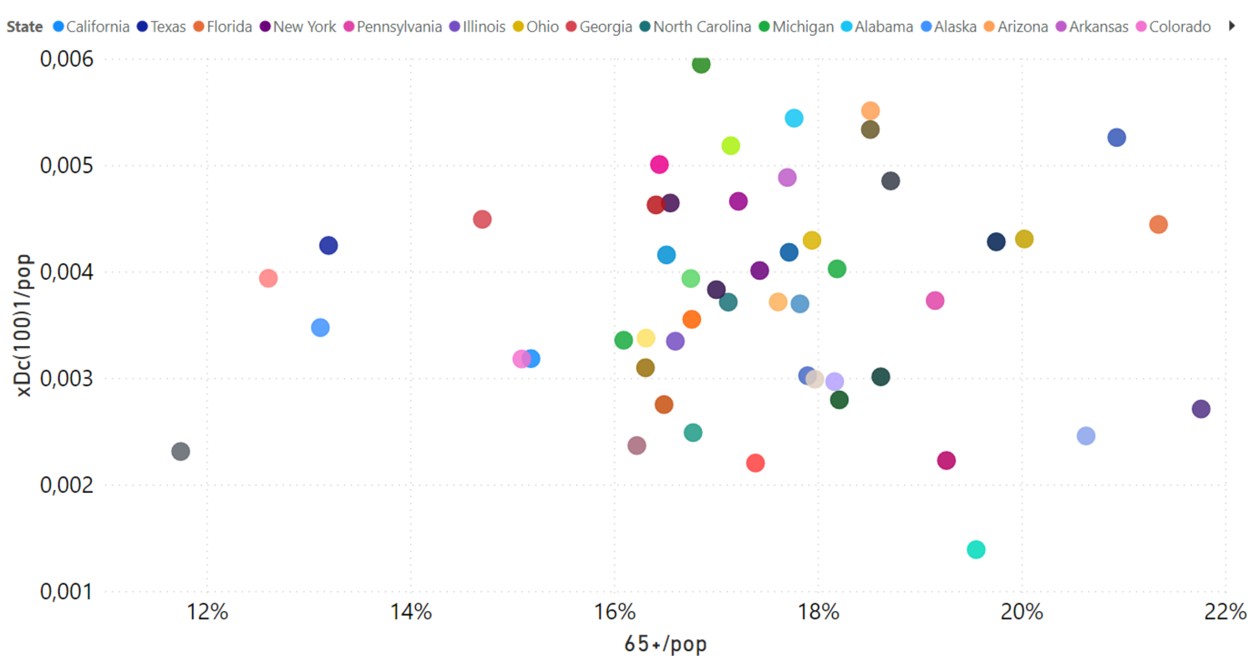


Figure 23. Excess mortality of the covid period normalized by population versus the proportion of people aged 65 and over in the USA. Each point is for one state of the USA. The color-code of the 51 states is shown in section 2. Data were retrieved as described in Table 1. xDc(100)1 is calculated from Equation 5.

There is no significant correlation (r = +0.046, “very weak”, Table 6). This is also true for the proportion of the population aged 75 years old and over (75+/pop) and for the proportion of the population aged 85 years old and over (85+/pop) (data not shown). Excess all-cause mortality of the 100-week covid period in the USA has no relation to old age, on a state-wise basis.

This lack of correlation with age again shows that the excess mortality is not consistent with having been caused by a viral respiratory disease, including COVID-19, since the known infection fatality ratios are exponential with age (Elo *et al.*, 2022; Sorensen *et al.*, 2022).

Other factors — which we did not consider in our previous article (Rancourt, Baudin and Mercier, 2021b) — are Supplemental Security Income (SSI) and Social Security Disability Insurance (SSDI). Those factors are state-provided benefits in case of disability or blindness (SSA, 2020). They can be interpreted as indicators or proxies for the proportion of frail populations in the USA. Whitaker (Whitaker, 2015) has interpreted that the majority of SSI and SSDI recipients can be classified as mentally disabled and receiving prescription psychiatric medication. He reports that some of these drugs are definitely associated with obesity. See also a current report about the prevalence of mental illness in the USA (NIMH, 2022).

Figure 24 shows the proportion of people receiving SSI versus the proportion of people receiving SSDI by state.

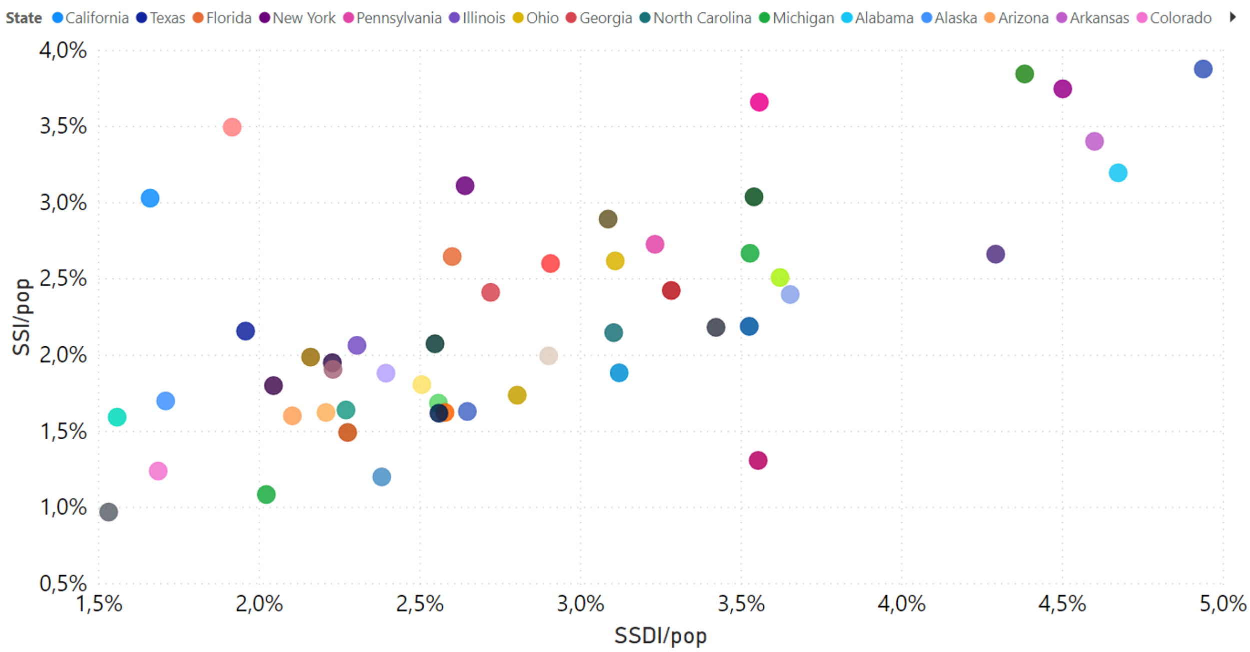


Figure 24. SSI recipients normalized by population versus SSDI recipients normalized by population in the USA. Each point is for one state of the USA. The color-code of the 51 states is shown in section 2. Data were retrieved as described in Table 1.

Although SSI and SSDI are independent programs, they are positively correlated to each other, showing that states that have more of one type of recipients also have more of the other type of recipients. Also, the two programs are not mutually exclusive, as some people called “concurrent” are eligible for both (SSA, 2020), and there is an approximately 10% overlap (data not shown).

Figure 25 shows the scatter plot for SSI recipients by population (SSA, 2022a). The Y-axis is the fraction xDc(100)1/pop, the 100-week covid-period excess mortality by population, which is the “100-week covid-period fatality ratio” for the USA population.

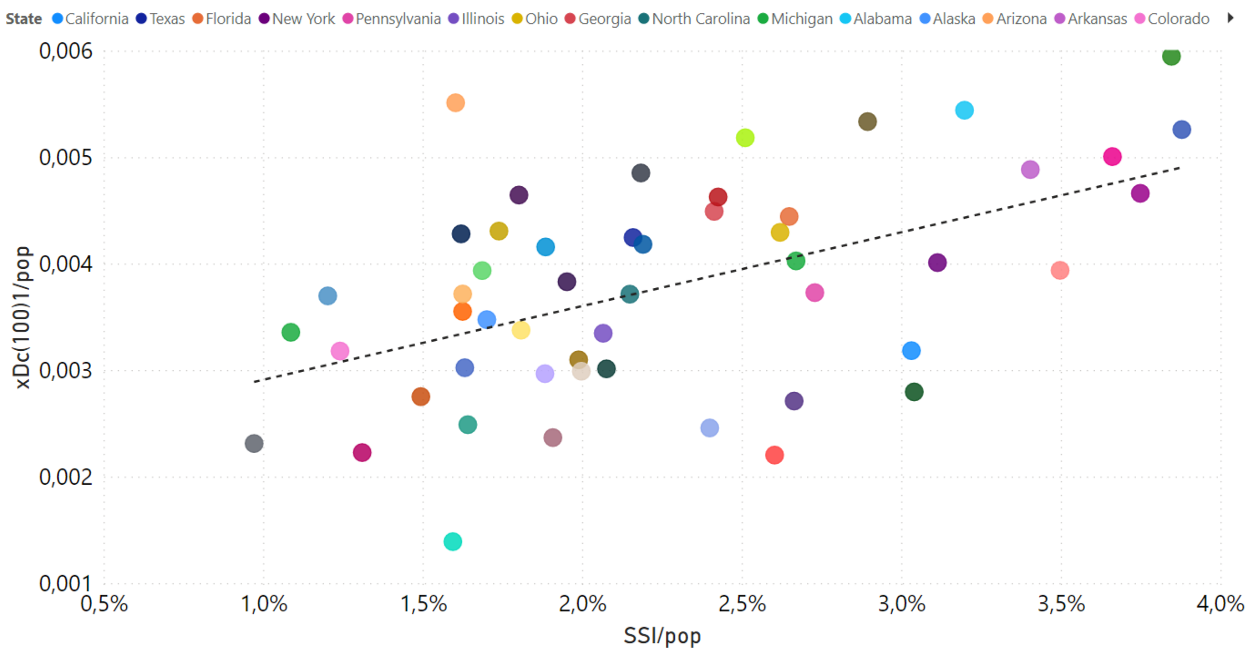


Figure 25. Excess mortality of the covid period normalized by population versus SSI recipients normalized by population in the USA. Each point is for one state of the USA. The parameters of the least squares fitted linear trend line are given in Table 6. The color-code of the 51 states is shown in section 2. Data were retrieved as described in Table 1. xDc(100)1 is calculated from Equation 5.

Here, the Pearson correlation coefficient is r = +0.51 ("moderate") (Table 6). The graph (Figure 25) suggests that a USA state with a SSI/pop of zero would nonetheless have a "100-week covid-period fatality ratio" of approximately 0.2%. This implies that the SSI population cannot account for all the excess mortality in the 100-week covid period: Other groups must also contribute to the said excess mortality.

Figure 26 shows the scatter plot for SSDI recipients by population, defined as the number of all disabled SSDI beneficiaries aged 18-64 (SSA, 2022b). The Y-axis is the fraction xDc(100)1/pop, the 100-week covid-period excess mortality by population, which is the "100-week covid-period fatality ratio" for the USA population.

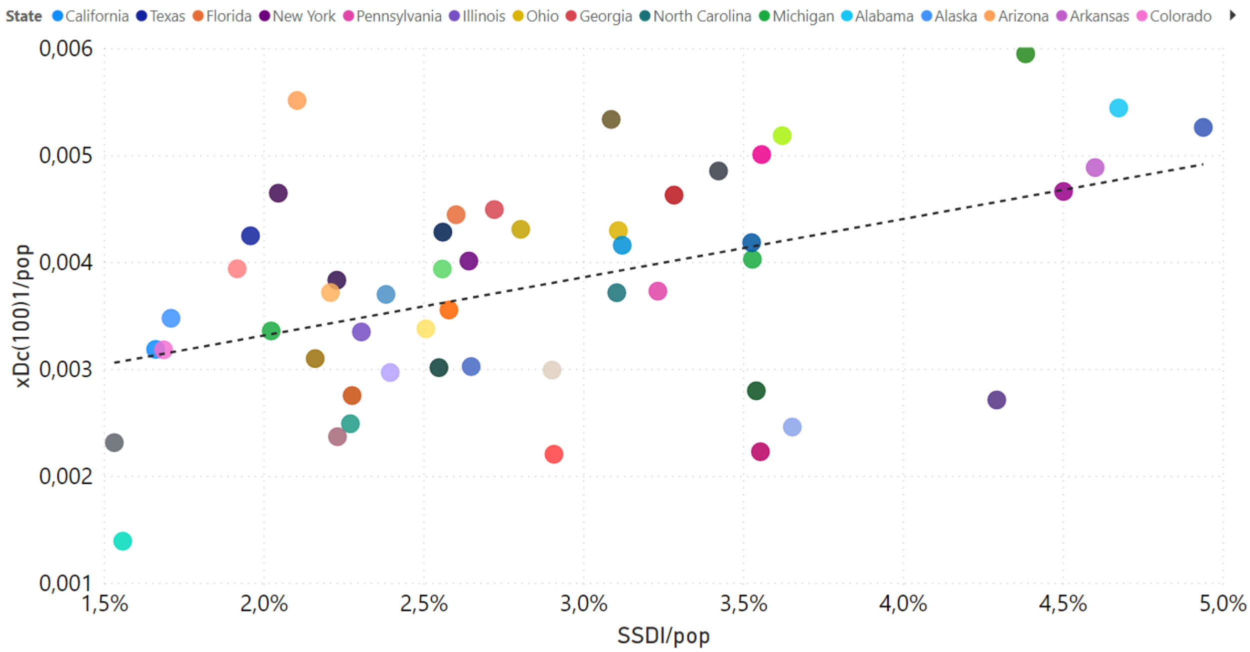


Figure 26. Excess mortality of the covid period normalized by population versus SSDI recipients normalized by population in the USA. Each point is for one state of the USA. The parameters of the least squares fitted linear trend line are given in Table 6. The color-code of the 51 states is shown in section 2. Data were retrieved as described in Table 1. xDc(100)1 is calculated from Equation 5.

Here, the Pearson correlation coefficient is r = +0.47 (“moderate”) (Table 6). The graph (Figure 26) suggests that a USA state with a SSDI/pop of zero would nonetheless have a “100-week covid-period fatality ratio” of approximately 0.2%. Like with the population of SSI recipients (Figure 25), this implies that the SSDI population cannot account for all the excess mortality in the 100-week covid period. Other groups must also contribute to the said excess mortality.

Figure 27 shows the scatter plot for disability, defined as the percentage of Americans living with a disability (Disabled World, 2020). Disability is defined as a long-lasting sensory, physical, mental, or emotional condition or conditions that make it difficult for a person to do functional or participatory activities such as seeing, hearing, walking, climbing stairs, learning, remembering, concentrating, dressing, bathing, going outside the home, or working at a job. The Y-axis is the fraction xDc(100)1/pop, the 100-week

covid-period excess mortality by population, which is the "100-week covid-period fatality ratio" for the USA population.

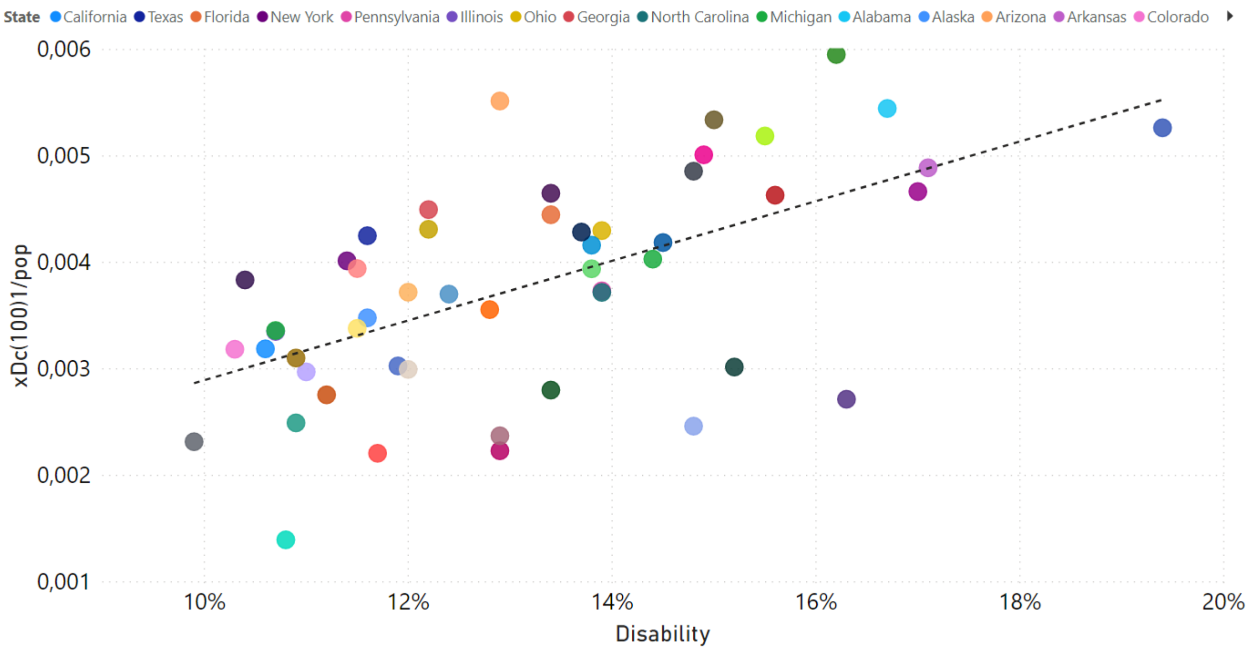


Figure 27. Excess mortality of the covid period normalized by population versus disability in the USA. Each point is for one state of the USA. The 8 apparent bottom outliers are: Hawaii, Massachusetts, New Hampshire, Washington, Rhode Island, Vermont, Oregon, and Maine. The color-code of the 51 states is shown in section 2. The parameters of the least squares fitted linear trend line are given in Table 6. Data were retrieved as described in Table 1. xDc(100)1 is calculated from Equation 5.

Here, the Pearson correlation coefficient is r = +0.59 ("moderate") (Table 6). The graph (Figure 27) suggests that a USA state with no one living with a disability would have a near-zero "100-week covid-period fatality ratio" (estimated at 0.01%). This is similar to the situation with poverty (Figure 20), in that if there were no disabled persons in the USA, the excess ACM of the covid period would have been essentially zero.

Table 6 gives parameters of the correlations discussed above.

Factor (units)	Slope (units)	Intercept	Pearson coefficient (r)	Strength (Evans, 1996)
Poverty (%)	+0.0331 (per %)	-0.00008	+0.855	Very strong
MHI ($)	-6E-08 (per $)	+0.008	-0.706	Strong
Obesity (%)	+0.0152 (per %)	-0.0011	+0.618	Strong
65+/pop (%)	+0.0023 (per %)	+0.0034	+0.046	Negligible to very weak
SSI/pop (%)	+0.069 (per %)	+0.0022	+0.512	Moderate
SSDI/pop (%)	+0.0546 (per %)	+0.0022	+0.466	Moderate
Disability (%)	+0.028 (per %)	+0.0001	+0.590	Moderate

Table 6. Parameters of the least squares fitted straight lines for xDc(100)1/pop (Y-axis) versus Factor (X-axis), where xDc(100)1/pop is dimensionless. Here: xDc(100)1/pop = Slope x Factor + Intercept.

In this article, we did not apply a strict separation between sections, which would exclude any discussion of results in the Results section, in order to facilitate appreciation for the often novel features of the data being presented.

In the next section, we continue, organize and supplement our discussion of the above results.

4. Discussion

4.1. All-cause mortality in the covid period in the USA: Sudden onset and heterogeneity by state

The covid period in the USA discontinuously starts immediately after the WHO's 11 March 2020 declaration of a pandemic, and is a period exhibiting extraordinarily large and time-wise (by week, by month, by season) anomalous ACM, compared to the historic record since at least 1999 (Figure 1). The sudden discontinuity is synchronous everywhere that it occurs, and its occurrence (presence and magnitude) is highly heterogeneous across state, provincial, regional and national jurisdictions, in North America and Europe, where the best ACM by time data is available (Rancourt, 2020; Rancourt, Baudin and Mercier, 2020, 2021a, 2021b; Johnson and Rancourt, 2022).

Such a large discontinuity, into a qualitatively different long-term (2-year) regime of ACM behaviour, has previously not been observed in epidemiology, so clearly. The break occurs between two regimes of ACM, between two distinct types of mortality behaviours by time, by age group and in terms of heterogeneity by jurisdiction, and it occurs at or near the date (11 March 2020) of the WHO's declaration of a pandemic; which is the date at which hospital, care-home and public health protocols were discontinuously, somewhat permanently and broadly changed, while lockdowns (jurisdiction-wide shelter-in-place or stay-at-home orders) were often and heterogeneously (by state) applied soon after this same date (Johnson and Rancourt, 2022), accompanied by massive restructuring of local economic activity.

Rancourt (Rancourt, 2020) seems to have been the first to point out this discontinuity in ACM by time and to have associated it to the measures installed on or near 11 March 2020, rather than to a pandemic spread of a contagious disease. We have discussed this break in detail previously, following Rancourt, and further associated it with the imposed structural changes in the society and the economy (Rancourt, Baudin and Mercier, 2020, 2021a, 2021b).

The heterogeneity by jurisdiction of the ACM by time behaviour following the said discontinuity is a striking phenomenon compared to remarkably uniform behaviour of ACM by time across jurisdictions, indeed across continents (at mid-latitudes), in pre-covid time (before 11 March 2020) (Rancourt, Baudin and Mercier, 2020). One has to go back to 1918 to observe a possibly similar phenomenon, at a time when less data was available (Rancourt, Baudin and Mercier, 2021b). In the USA, there are particularly large state-to-state differences in ACM by time behaviour during the covid period, compared to very similar state-to-state behaviour in pre-covid time (Rancourt, Baudin and Mercier, 2021b). For example, Johnson and Rancourt (Johnson and Rancourt, 2022) find covid-period health-status-adjusted integrated ACM per capita to vary by approximately 20% from state to state for the covid period, while a state-to-state variation of only approximately 2% occurs for corresponding integration windows prior to 11 March 2020 (their Figure 7).

The USA state-wise heterogeneity in ACM behaviour is a further demonstration of the abrupt change in ACM regime that occurred on or near 11 March 2020. Given the complexities of the comparative behaviours between states, there is no substitute for showing the all-ages data for each of the states. This is done for the ACM/w data in Appendix A.

We previously showed that in the USA the ACM by time and by state jurisdiction in the covid period is contrary to the expected behaviour for a viral respiratory disease pandemic, and that the extra deaths, when and where they occur in the USA, were likely due to the government and medical responses, including constructive denial of treatment of an unprecedented bacterial pneumonia epidemic that predominantly affected poor and obese individuals living in hot-climate states (Rancourt, Baudin and Mercier, 2021b).

More specifically, we proposed the following interpretive scheme:

- The covid response and measures created stressful socio-economic, regulatory and institutional conditions. For example, studies report increased unemployment

and worsening mental health (Czeisler *et al.*, 2020; Jewell *et al.*, 2020; Giuntella *et al.*, 2021). This would result in chronic psychological stress in many individuals, during the covid period.

- As we have discussed and reviewed previously (Rancourt, Baudin and Mercier, 2021a), chronic stress debilitates the immune system and is arguably the dominant determinant of individual health (Cohen, Tyrrell and Smith, 1991; Ader and Cohen, 1993; Cohen *et al.*, 1997; Sapolsky, 2005; Cohen, Janicki-Deverts and Miller, 2007; Dhabhar, 2014; Prenderville *et al.*, 2015). Furthermore, the molecular and physiological mechanisms for suppression of the immune system by experienced chronic stress are being elucidated more and more (Devi *et al.*, 2021; Udit, Blake and Chiu, 2022).
- In terms of assigning actual cause of death for covid-period excess mortality in the USA, we argued that bacterial pneumonia was a likely candidate, attacking vulnerable groups subjected to debilitating stress, during a massive pneumonia epidemic evident in the CDC data, combined with a dramatic drop in antibiotic prescriptions (Rancourt, Baudin and Mercier, 2021b). The said pneumonia epidemic is also seen, directly or indirectly, in other studies (Di Gennaro *et al.*, 2021; Bradley *et al.*, 2022).
- Further studies have since established a sustained drop in antibiotic prescriptions (e.g., (Buehrle *et al.*, 2021; King *et al.*, 2021; Kitano *et al.*, 2021; Van Laethem *et al.*, 2021, 2022; Gisselsson-Solen and Hermansson, 2022; Givon-Lavi *et al.*, 2022; Gottesman *et al.*, 2022; Knight *et al.*, 2022; Winglee *et al.*, 2022).

Those conclusions are supported by the present study, which has the added benefits of:

- month-wise time-resolved ACM by age group and by sex back to 1999;
- more recent consolidated week-wise time-resolved ACM, up to and including week-5 of 2022;
- closer examination by age group; and
- cumulative vaccine dose delivery data time-resolved by week, by injection series or status, by age group and by state.

In particular, the correlations between xDc(100)1/pop (the 100-week covid-period excess mortality by population, which is the “100-week covid-period fatality ratio” for the USA population) and poverty (Figure 20), median household income (MHI, Figure 21), obesity (Figure 22), SSI/pop (SSI recipients per population) (Figure 25), SSDI/pop (SSDI recipients per population) (Figure 26), disability (Figure 27), and the absence of significant correlations with population fractions of elderly residents (Figure 23, and above discussion) (Table 6), provide compelling support for the said conclusions. For example, the absence of significant correlations with population fractions of elderly residents (65+, 75+, or 85+ years) is incompatible with the reported exponential age-dependence of the COVID-19 infection fatality ratio (Elo *et al.*, 2022; Sorensen *et al.*, 2022), and contrary to all the studies finding that the dominant factors are age and age-associated comorbidities for viral respiratory diseases, including COVID-19. Whereas, no known respiratory-disease virus specifically targets residents living in poverty (Figure 20), irrespective of age (Figure 23).

4.2. Late-summer-2021 anomalous mortality of young adults

The time-structure of the all-cause mortality by month (ACM/m) from 2000, into the covid period, by age group is shown in Figure 4. Here, the relative magnitude of the covid-period excess mortality above the historic trend is particularly large for the age groups 25-34y (Figure 4C), 35-44y (Figure 4D), and 45-54y (Figure 4E).

See also Figure 7, Figure 9, Figure 10, Figure 11, Figure 13, Figure 15, Figure 17 and Figure 19. Basically, we observe the same age-group-differential and seasonal-differential ACM by time phenomena with higher time resolution and in more detail in the all-cause mortality by week (ACM/w).

Similarly with the ACM/m (Figure 4) data, and as is evident from Table 3, the ACM/w (Figure 7) data also shows that the relative magnitude of the covid-period extra deaths above the historic trend is particularly large for the age group 25-44y (Figure 7B), and to a lesser degree 45-64y (Figure 7C), especially the late-summer-2021 feature (smp2).

These covid-period young-adult age group large excesses in ACM by time, especially in the late-summer-2021 (smp2) feature, are a central feature of mortality during the covid period in the USA.

The age-group-dependent relative magnitude of the covid-period excess mortality is contrary to the age dependence of mortality for viral respiratory diseases, including that reported for COVID-19, in which mortality strongly increases exponentially or near-exponentially with age (Elo *et al.*, 2022; Sorensen *et al.*, 2022).

These results are contrary to, incompatible with, and irreconcilable with an interpretation in which excess mortality (by age-group) in the covid period in the USA is mostly or predominantly caused by COVID-19; or any known viral respiratory disease (see Rancourt et al.'s discussion about the 1918 declared pandemic (Rancourt, Baudin and Mercier, 2021b), and references therein). Either one must admit that the declared COVID-19 pandemic is not the main cause of death to explain the excess mortality data, or ignore the well-established data showing that COVID-19-assigned mortality increases exponentially or near-exponentially with age, and that young people essentially (comparatively) do not die from COVID-19, as the primary assigned cause of death in a controlled clinical and laboratory verified setting.

Furthermore, relative mortality is particularly large for the late-summer-2021 feature (smp2) in the 35-44y age group (Figure 4D), compared to any other time in the covid period, and more so than with any other age group. This feature (large smp2 in the 35-44y age group), however, is highly variable from state to state, being prominent or very prominent in states such as Texas, Florida, Georgia, North Carolina, South Carolina, Alabama, Arkansas, Hawaii, Idaho, Kentucky, Louisiana, Mississippi, Missouri, Nevada, Oklahoma, Oregon, Tennessee, Washington, Virginia, West Virginia and Wyoming, while being absent in New York and New Jersey, intermediate in California, and mostly intermediate or absent in other states, while Michigan uniquely has a spring-2021 peak in mortality for that age group centered in April (Figure 11). The latter observations are confirmed in the ACM/w data for 25-64y age group (not shown). Generally, the 2020

and 2021 summers were most deadly in the Southern states, as previously described (Rancourt, Baudin and Mercier, 2021b). Such state-to-state heterogeneity is inconsistent with the pandemic paradigm of rapid spread, extensive coverage and complete immune susceptibility. It is more understandable in terms of the driving forces described above.

Coming back to age-groups: Why would this be? Why would mortality in this young-adult age group suddenly spike in late-summer-2021, in many states and as seen on the basis of the whole USA, to an unprecedented large value, after 18 months of the declared pandemic, compared to anything in the earlier covid period or the last 20 years, approximately doubling all-cause mortality for several months for 35-44 year olds (Figure 4D), both male and female (not shown)? See also Figure 7, Figure 9, Figure 10, Figure 11, Figure 13, Figure 15, Figure 17 and Figure 19.

In attempting to answer this question (Why are young adults dying more than ever in the second half of the covid period, and in the late-summer-2021 ACM peak in particular?), we submit that the answer is probably not "variants of concern", or any such theoretical proposal from immunology. Instead, we describe two preferred hypotheses to explain the observation:

i. The first is that young adults are more resilient than old adults against the cumulative impact of persistent covid-period conditions that cause chronic psychological stress that, in vulnerable groups, causes emergent or worsening immunodeficiency that enables death by bacterial pneumonia. In support of this hypothesis, one of the largest vulnerable groups in the USA — those afflicted by serious mental illness (5.6% adults = 14.2 million aged 18+, in 2020) — has a heavily skewed prevalence towards young adults (see below).
ii. The second is that the vaccination campaign, including the "vaccine equity" campaigns, captured many thus made immunocompromised young adults from vulnerable groups and that the vaccine challenge against many of these individuals constituted a significant comorbidity, which was absent in the first

(pre-vaccination) half of the covid period, thus increasing the death toll of young adults, overall, in the second (vaccination) half of the covid period.

Note that the second hypothesis (vaccine toxicity) relies on the conditions described in the first hypothesis (cumulative stress-induced immunodeficiency). This is because the vaccine toxicity for subjects who are not immunocompromised (fatality risk per dose, inferred from VAERS data) is too small to quantitatively explain the observed ACM increases that are synchronous with increases in vaccine-delivery (administered doses), assuming avoidance of immunocompromised subjects (see above, and below).

As mentioned above, we do not believe that any “variant of concern” (CDC, 2022e) emerging in 2021 could produce such a result in the mortality data, or that the explanation is viral. Rather, we prefer to propose that the same forces that appear to generally determine the exceptionally large excess mortality in the covid period in the USA — namely the impact on the immune systems of individuals in populations of those most vulnerable to psychological stress and social isolation during life-changing covid-period circumstances, combined with an essentially untreated mass bacterial-pneumonia epidemic (Rancourt, Baudin and Mercier, 2021b) — also largely determined the jurisdictional, age and time structures of excess mortality in the covid period, on the background of the demographics of highly vulnerable groups. Here, the hypothesis is that, while the “conditions = stress = immune-vulnerability = death from pneumonia” scenario existed from the very start of the covid period (Rancourt, Baudin and Mercier, 2021b), the prolonged conditions and associated chronic stress eventually has more relative impact on young adults that are more resilient at first, thus changing the age structure of mortality as the covid period advances.

We expect, therefore, that the change in age structure of mortality during the course of the covid period (Figure 15, Figure 17) is driven by such factors as a “dry tinder effect” among the elderly and differential youth resilience to chronic stressors (relative endurance over long periods), on the background of the demographics of highly vulnerable groups, rather than driven by the vaccination campaign *via* general-

population vaccine toxicity (fatality risk per dose for non-immunocompromised subjects) acting alone and irrespective of these circumstances.

There is also a cumulative effect on young adults, which is irreversible to some extent (Giuntella *et al.*, 2021). Our interpretation of Figure 15 is consistent with the fact that the hardships (expenses, housing and food insecurity) are sustained in the USA during the covid period (CBPP, 2022). In the words of the OECD (OECD, 2022):

> "The COVID-19 pandemic has triggered one of the worst jobs crises since the Great Depression. There is a real danger that the crisis will increase poverty and widen inequalities, with the impact felt for years to come."

Also, socio-economic factors may have caused young adults to have higher experienced stress in the second half of the covid period, compared to older adults. For example, pressures inducing bankruptcies and associated losses of livelihood and personal identity would increase as the restrictive conditions persist in many sectors, although analysis of the macroeconomic data is complex (Martos-Vila and Shi, 2022).

It is also possible that the age-structure change phenomenon partly results from or is significantly contributed to by vaccine-campaign (including so-called "vaccine equity" campaigns) capture of vulnerable young adults made immunocompromised by the said chronic psychological stress.

Both of the latter hypotheses (relative resilience to stress of young adults in vulnerable groups and vaccine capture of young adults made immunocompromised by chronic stress) advanced to explain the increased skewness of mortality towards young adults in the vaccination period (second half) of the covid period are consistent with the fact that the prevalence of serious mental illness is large and highly skewed towards young adults in the USA (NIMH, 2022). Indeed, the age distribution in covid-period fatality risk that we observe, which is skewed towards young adults in the vaccination period of the covid period (Figure 17), should be put in the context of the prevalence of serious mental illness, which was 14.2 million adults aged 18 or older in the USA in 2020,

representing 5.6% of all USA adults, and which is highly skewed towards young adults (NIMH, 2022):

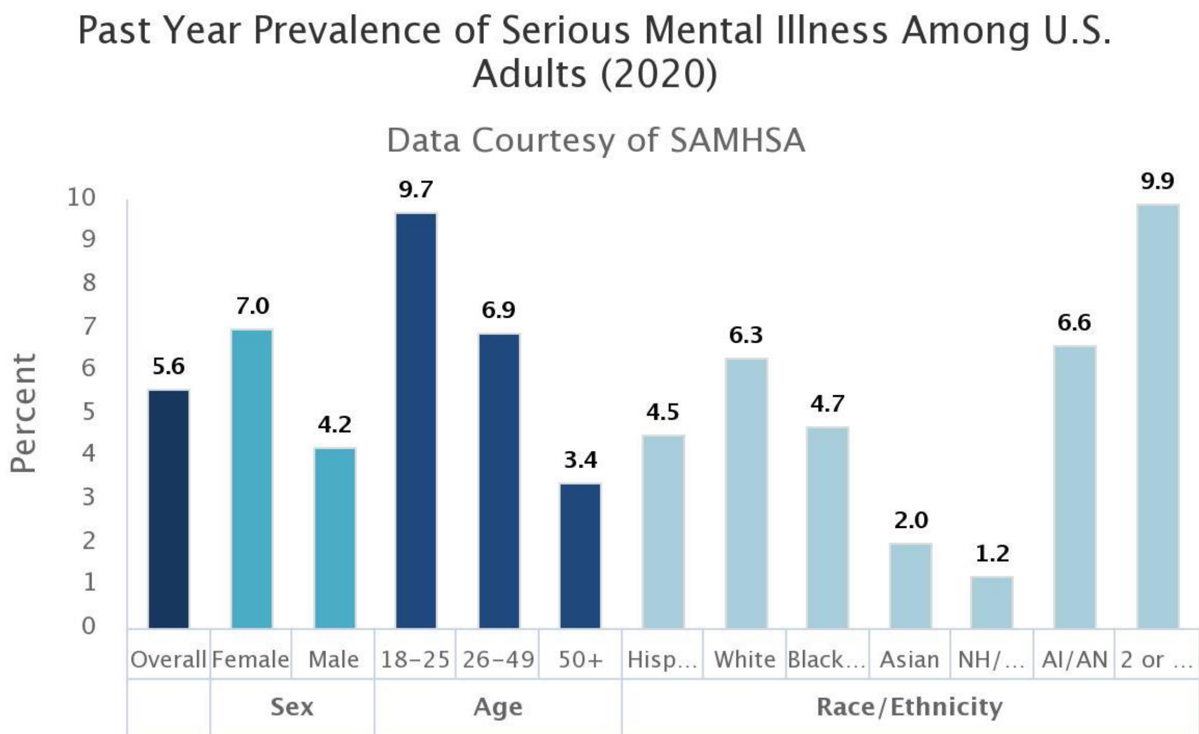


Figure 28. Prevalence of serious mental illness among U.S. adults in 2020. Data are shown for the entire USA (Overall), by sex (Female, Male), by age (18-25, 26-49, 50+) and by race/ethnicity (Hispanic, White, Black or African American, Asian, Native Hawaiian/Other Pacific Islander, American Indian/Alaskan Native, Two or more races). Serious mental illness is defined as a mental, behavioral, or emotional disorder resulting in serious functional impairment, which substantially interferes with or limits one or more major life activities. This figure is from NIMH (NIMH, 2022).

Basically, any model of excess mortality, which relies on the mentally disabled as a source group and serious mental illness as a major cofactor, will be biased towards mortality risk that is skewed towards young adults. It is well established that in the USA younger people are disproportionately affected by diagnosed mental disorders (Merikangas *et al.*, 2010).

We advance that the tragic excess deaths of 35-44 year olds (and 25-64 year olds) in late-summer-2021 in the USA, extraordinarily exhibited as an actual peak (smp2) in

ACM by time, for example, needs to be explained by specific health-status and socio-psycho-economic circumstances in the different jurisdictions, and not solely in terms of theoretical proposals from virology and immunology (e.g., "variants of concern", etc.). The needed actual community-level field work is not being sufficiently funded or undertaken, to our knowledge.

4.3. Vaccination campaign

The time-resolved and age-group resolved vaccination campaign, together with the similarly resolved ACM/w show that the vaccination campaign did not reduce mortality during the covid period (Figure 10; Figure 11; Figure 12; Figure 13; Figure 14; Figure 16; Figure 17; Table 4; Table 5).

We conclude with a high degree of certainty that the COVID-19 vaccination campaign in the USA was ineffective in reducing all-cause mortality. The mass vaccination campaign was not justified in terms of reducing excess all-cause mortality. The large excess mortality of the covid period, far above the historic trend, was maintained irrespective of the unprecedented vaccination campaign.

Furthermore, the vaccination campaign may have affected the age structure of ACM by contributing to the deaths of young adults in vulnerable groups but the same dominant forces that caused the large excess ACM in the first (50-week, pre-vaccination) half of the covid period appear to have continued to cause the large excess ACM in the second (50-week, vaccination) half of the covid period.

In the ACM/w data (Figure 7), similarly to the ACM/m data (Figure 4), relative mortality is particularly large for the late-summer-2021 feature (smp2) in the 25-44y age group (Figure 7B), compared to any other time in the covid period, and more so than with any other age group. It is also anomalously large, to a lesser degree, for the age group 45-64y (Figure 7C).

This feature in ACM by time during the covid period (an exceptionally large late-summer-2021 peak, smp2) is highly variable from state to state (see above, Figure 11, and see Appendix A), and occurs after the majority of the main-series vaccination campaign has been mostly completed.

Nonetheless, is some or most of the exceptional mortality occurring in the late-summer-2021 period (smp2) consistent with having been caused by the vaccination campaign? Likewise, is any feature in ACM by time consistent with having been caused by the vaccination campaign?

Figure 10 allows a direct comparison, on the same time axis, of all-cause mortality by week and cumulative number of vaccinated individuals, by vaccine sequence (1st dose, fully vaccinated, booster), for separate age groups. Figure 11 allows the same for specific states.

A study of the Vaccine Adverse Event Reporting System (VAERS) data of the USA has shown that the deaths associated with the COVID-19 vaccine in the USA typically occur first in a large initial peak within 5 days or less following the injection; followed (~5 days to ~60 days post injection) by a shoulder of exponential decay in deaths, with a fitted half-life decay time typically in the range 13-30 days (Hickey and Rancourt, 2022; their figures S3 through S5).

This means that deaths associated with the injections in the USA occur essentially immediately following delivery of the injection (mostly within days, with a decaying residual risk of fatality lasting weeks).

In addition, it is usually postulated that the alleged life-saving benefits of the vaccine become operative 7-14 days from the time of injection, and should last several months, similarly to the 90 days or so of efficacy claimed for flu vaccines (Rambhia and Rambhia, 2019).

In this way, any measurable positive or negative impact of the vaccination campaign on death rate (all-cause mortality by week) should be temporally associated with times of large or maximum slope in cumulative vaccine dose delivery (or vaccinated status acquisition), if vaccine fatality toxicity is large enough (deleterious impact) or vaccine protection against death is large enough (positive impact).

An increase in mortality from the vaccination campaign would be seen within 5 days or so of a large slope in cumulative vaccine dose delivery, whereas a smaller mortality would be seen to follow a large slope in cumulative vaccine dose delivery (or vaccinated status acquisition) by a few weeks or more and should be persistent after having attained significant vaccine dose coverage.

As discussed above in presenting Figure 10 and Figure 11, there is a modest but significant stepwise increase in 1st-dose vaccine delivery (administration), which is synchronous with the late-summer-2021 peak in ACM, visible for all ages and for the 25-44 and 45-64 years age groups (Figure 10A, C, D). This temporal association is prominent in the data for many specific states (e.g., Figure 11), and cannot be dismissed as noise.

We estimate that, in order to achieve quantitative agreement between the outcome (late-summer-2021 peak integrated excess mortality) and factor (additional vaccine doses over the period of occurrence of the peak), the vaccine adverse-effect fatality toxicity per dose would need to be approximately 100 times the estimated non-immunocompromised fatality toxicity per dose (Hickey and Rancourt, 2022; their Table 1), assuming that only non-immunocompromised resident were injected. There are many more doses administered than deaths. This means that if immunocompromised residents from vulnerable groups were captured in the vaccination doses delivered in the relevant period, then it is possible that the late-summer-2021 mortality peak is entirely or partly due to vaccine challenge of vulnerable young adults.

In this regard, it is relevant that the so-called “vaccine equity” campaigns in the USA were operating in the relevant period:

- A *JAMA* Editorial of 29 January 2021, entitled “Vaccine Distribution—Equity Left Behind?” recommended, among other things “1. Prioritize vaccine distribution to zip codes that have been most severely affected by COVID-19 and that have high indexes of economic hardship”, and so on (Jean-Jacques and Bauchner, 2021).
- The *New York Times* provided extended reporting on county-wise vaccine coverage (*The New York Times*, 2022).
- Large foundations such as the Rand Corporation were significantly involved supporting “vaccine equity” programs (Faherty *et al.*, 2022).
- Louisiana, for example, had fully launched its comprehensive “vaccine equity” program, as did virtually all states to varying degrees (*Louisiana Launches Grassroots COVID Vaccine Campaign to Ensure No Community Gets Left Behind* | *Office of Governor John Bel Edwards*, 2021).

Similarly, as discussed above in introducing Figure 11, Michigan has a unique feature in its ACM by time data, not seen for any other state. Michigan has a unique April-2021-centered spring-2021 peak in ACM for young adults, which coincides with the large main onset of the vaccination campaign for these ages (Figure 11G, H). In this case, in order to achieve quantitative agreement between the outcome (spring-2021 peak integrated excess mortality) and factor (additional vaccine doses over the period of occurrence of the peak), the vaccine adverse-effect fatality toxicity per dose would need to be approximately 10 times the estimated non-immunocompromised fatality toxicity per dose (Hickey and Rancourt, 2022; their Table 1), assuming that only non-immunocompromised resident were injected. There are many more doses administered than deaths. This means that if immunocompromised residents of Michigan from vulnerable groups were captured in the vaccination doses delivered in the relevant period, then it is possible that the unique spring-2021 mortality peak for Michigan is entirely due to vaccine challenge of vulnerable young adults. We consider this to be the most likely hypothesis we can make, with the available information, to explain the

unique spring-2021 mortality peak for Michigan. If the hypothesis is correct, then this demonstrates the principle that vaccine challenge of residents made immunocompromised by chronic stress can explain large features in ACM by time, in the covid-period and vaccine-campaign circumstances.

4.4. Looking ahead

Unavoidable questions are: “When will the covid period end?” and “Will ACM by time and by jurisdiction return to the pre-covid-period normal?”

We quickly looked at the latest ACM/w data for the USA, which appears to be reliable through to April-2022, in order to give tentative answers, looking forward.

The data (shown in Appendix C) has the 2021-2022 winter peak in ACM dropping precipitously in February-2022, down to a level, in March and April 2022, which is typical of pre-covid-period summer baseline values. Such a low value did not occur at any time in the USA in the covid period that we studied in the present article.

It would seem that, in terms of all-cause mortality, the covid period ended, at least momentarily, in March and April 2022. It will be interesting to see whether there will be a summer-2022 peak in ACM when more data becomes available.

Late reporting of mortality to the CDC could alter the above tentative observation.

5. Conclusion

Our results show the following overall large-scale features:

- All-cause mortality by time in the USA is heterogeneous by state and persistently far in excess of the recent historic decadal trend, starting immediately when a pandemic was declared by the WHO on 11 March 2020, and continuing

throughout the entire covid period that we examined, up to the week ending on February 5, 2022 — with a total of 1.27M excess deaths (Figure 1, Figure 3, Figure 5, Figure 6; Table 2, Table 3).

- Throughout the covid period, all-cause mortality is heterogeneous by state and anomalous in its time (by week, by month) and seasonal variations, compared to historic behaviour. The anomalies include winter and summer peaks, which are highly variable in magnitude from year to year in the covid period, and from state to state (Figure 8, Figure 9; Appendix A); as we observed previously (Rancourt, Baudin and Mercier, 2021b). The broad "summer peaks" of ACM by time in 2020 and 2021 are of a nature that has not previously been observed in mortality data for the USA or any country, historically, since quality data has been available for more than 100 years. The anomalous heterogeneity by state in integrated mortality over the covid period was recently demonstrated by Johnson and Rancourt (Johnson and Rancourt, 2022; their Figure 7).

- Unlike for viral respiratory diseases, including the presumed SARS-CoV-2 virus itself (Elo *et al.*, 2022; Sorensen *et al.*, 2022), the covid-period excess mortality risk by age group is not predominantly confined to the elderly population; and the inferred age-group-specific infection fatality ratios are not exponential or near-exponential with age, as they would be (Table 2, Table 3, Figure 4, Figure 7, Figure 9). On the contrary, overall for the covid period, mortality risk is broadly distributed to all age groups and is significantly larger for younger adults compared to the eldest adults (Figure 9). The non-exponential-with-age (more age-uniform) distribution of mortality risk to all age groups holds for both the first half (pre-vaccination) and second half (vaccination) of the 100-week covid period (Figure 10, Figure 13, Figure 15, Figure 17, Table 4). The observed age-group distribution of all-cause mortality risk constitutes proof that the covid-period excess mortality cannot predominantly be due to the presumed SARS-CoV-2 virus or to any viral respiratory disease. The alternative would be to abandon the accepted body of research on mortality risk by age.

- Instead of the covid-period excess all-cause mortality risk being predominantly (or even moderately) determined by age of the population, the state-wise integrated excess all-cause mortality for the entire 100-week covid period normalized by state population (outcome) is correlated to socio-economic factors that are macro-indicators of state-wise resident vulnerability (Table 6):
    - The covid-period excess all-cause mortality risk is very strongly correlated to poverty (r = +0.86) (Figure 20).
    - The said mortality risk is strongly correlated to MHI (Median Household Income) (r = -0.71) (Figure 21).
    - The said mortality risk is strongly correlated to obesity (r = +0.62) (Figure 22).
    - The said mortality risk is not correlated simply to age of the population. This is shown for 65+ ages in Figure 23, and is maintained for 75+ and 85+ ages (not shown).
    - The said mortality risk is moderately correlated to the number of SSI (Supplemental Security Income) recipients by population (r = +0.51) (Figure 25).
    - The said mortality risk is moderately correlated to the number of SSDI (Social Security Disability Insurance) recipients by population (r = +0.47) (Figure 26).
    - Whitaker (Whitaker, 2015) has interpreted that the majority of SSI and SSDI recipients can be classified as mentally disabled and receiving prescription psychiatric medication.
    - The said mortality risk is moderately correlated to disability (r = +0.59) (Figure 27).

- Despite the fact that there are significant changes in age structure of ACM by time during the course of the covid period, the overall qualitative behaviour of ACM by time (anomalously large excess mortality and presence of anomalous summer and winter seasonal variations in ACM by time) and the 50-week-integrated excess ACM are not substantially different in the first half of the

100-week covid period (first 50 weeks of the covid period), in which there was essentially no vaccination campaign, and in the second half of the 100-week covid period (second 50 weeks of the covid period), in which most of the vaccination campaign was accomplished (Figure 4, Figure 7, Figure 13 , Figure 15, Figure 17, Figure 19). Therefore, the suddenly applied and massive vaccination campaign (Figure 10) did not induce a large change of regime from one type of ACM by time to another, on the scale of the dramatic change in regime from pre-covid to covid period.

- Regarding mortality averted by vaccination, the COVID-19 vaccination campaign in the USA did not cause any seasonally unambiguous temporally associated decrease in all-cause mortality, for all ages or in any age group (Figure 10; see also Figure 11). The vaccination campaign did not measurably cause any deaths to be averted. This is contrary to the notion that the vaccines are “effective” in reducing “serious illness” (and presumably death), becoming operative 7-14 days following the time of injection, with the protection presumably lasting at least several months.

- Therefore, although much messaging attention is directed towards life-saving consequences arising from the mass vaccination campaign in the USA, clearly such effects are both undetectable in all-cause mortality and necessarily small compared to the overwhelming harm from the extraordinary covid-period conditions themselves.

- Conversely, regarding vaccine-induced mortality, the COVID-19 vaccination campaign in the USA did not cause the 50-week-integrated excess ACM in the second half of the 100-week covid period (second 50 weeks of the covid period), in which most of the vaccination campaign was accomplished, to be systematically larger (systematically across all age groups, or all states) than in the first half of the 100-week covid period (first 50 weeks of the covid period), in which there was essentially no vaccination campaign. The persistent socio-economic, regulatory, institutional… changes associated with the covid period

(relative to pre-covid behaviour) had a large effect compared to changes associated specifically with the period of the vaccination campaign, positive or negative (Figure 4, Figure 7, Figure 13 , Figure 15, Figure 17, Figure 19).

- Despite the fact that there is no large systematic effect of the vaccination campaign on either 50-week-integrated mortality or main qualitative features of ACM by time, positive or negative, we nonetheless detect significant seasonally unambiguous local temporal associations between increases in number of vaccinated residents and synchronous increases in all-cause mortality, for certain age groups, and most prominently in certain states:
    - The largest of these local temporal associations is seen in the data for the whole USA and all age groups, as an accelerated increase in cumulative number of residents having received at least one dose (or being fully vaccinated), which is synchronous with the late-summer-2021 surge in ACM by time (Figure 10A).
    - The said local temporal association is most evident for the 25-44 years age group (Figure 10C), also prominent for the 45-64 years age group (Figure 10D), and discernible for the 65-74 years age group (Figure 10E).
    - The said local temporal association is most prominent for the 25-64 years age group in Southern states — which typically have the smallest vaccination rates — including: Florida, Georgia, Louisiana, Mississippi and Alabama (Figure 11).
    - The special case of Michigan is also noteworthy (Figure 11G, H), as discussed above.

The latter observations lead us to conclude that the large changes in age structure of ACM by time (first half versus second half of the covid period) (esp. Figure 17) may be partly (see Discussion section) or largely due to aggressive “vaccine equity” campaigns that captured immunocompromised young adults in Southern states, thus causing disproportionate mortality among vulnerable young adults in late-summer-2021.

The entire picture of mortality during the covid period in the USA, which included implementation of the vaccination campaign after the first 50 weeks or so, can be modelled as:

covid-period socio-economic, regulatory, institutional… conditions

⟶ psychological stress / social isolation

⟶ severely suppressed immune system in most vulnerable residents (+ vaccine assault of thus immunocompromised vulnerable residents)

⟶ mortality from untreated bacterial pneumonia (+ vaccine-assault comorbidity) in most vulnerable residents

The model arises as follows.

- We infer from the temporal and jurisdictional characteristics of age-group-resolved excess ACM that large structural changes in the living and care conditions of residents of the USA — directly enacted by state and institutional players (including employers) during the covid period and including secondary consequences of the said directly enacted changes — are causally associated with the large and sustained excess mortality in the covid period.
- We infer from correlations with socio-economic factors that severe harm and death were induced by the said covid-period changes in particular classes of residents, such as isolated, sick, disabled, dependent, obese, poor, seriously mentally ill or elderly individuals; and of course residents who are co-afflicted by such conditions.

- We postulate that the mechanistic connection between the said covid-period changes and high risk of all-cause death in vulnerable residents is the well-established link between experienced psychological stress and social isolation (factor) and suppressed immunity, ill-health and death (outcome).
- We postulate that the end-point mechanistic cause of death in the thus immunocompromised vulnerable groups is bacterial pneumonia, in the midst of a recorded mass epidemic of bacterial pneumonia, at a time when antibiotic prescription rates showed an unprecedented decrease, in addition to aggressive vaccine challenge ("vaccine equity" programs) in late-summer-2021.

The model is developed and contextualized in more detail in the Results and Discussion sections. It provides a plausible and consistent explanation for all the aspects of the ACM data for the USA, including the large change in age structure of the ACM on entering the vaccination-campaign part of the covid period.

The model is predictive in that any type of comparable sudden socio-economic upheaval, such as war or a Great Depression, in societies with large pools of vulnerable residents, would give rise to this kind of large and rapid increase of mortality, targeting the most vulnerable, with bacterial pneumonia playing a major role. We have previously advanced that 1918 was such an episode in mid-latitude nations (Rancourt, Baudin and Mercier, 2021b).

In conclusion, in terms of all-cause mortality, the covid-period socio-economic, regulatory, institutional… conditions in the USA (from 11 March 2020 to week-5 of 2022) were in-effect a large-scale deadly assault against vulnerable groups, which killed approximately 1.27M members of the said groups. The temporal, jurisdictional and age-group characteristics of the mortality are incompatible with the excess mortality having been primarily caused by the presumed SARS-CoV-2 viral respiratory disease virus. In the absence of poverty or if the covid-period socio-economic, regulatory, institutional… conditions had not been imposed, there most probably would not have been excess mortality in the USA, which was essentially the case in neighbouring Canada (Rancourt,

Baudin and Mercier, 2021b; their Section 4). The COVID-19 vaccination campaign, accomplished in the second half of the covid period, did not avert any deaths, and may have been a significant contributing factor causing excess mortality in vulnerable-group young adults during late-summer-2021.

In regard to the fundamental results of this study, we would recommend a transparent and accountable large-scale state, county and community level independent forensic investigation of the deaths, excluding the involvement of interested government agencies and private corporations. The mandate should include broad systemic considerations, in addition to specific circumstances, and the investigators should have the necessary powers and resources consistent with the magnitude and extent of the catastrophe, in the hope of preventing any similar public health disaster in the future.

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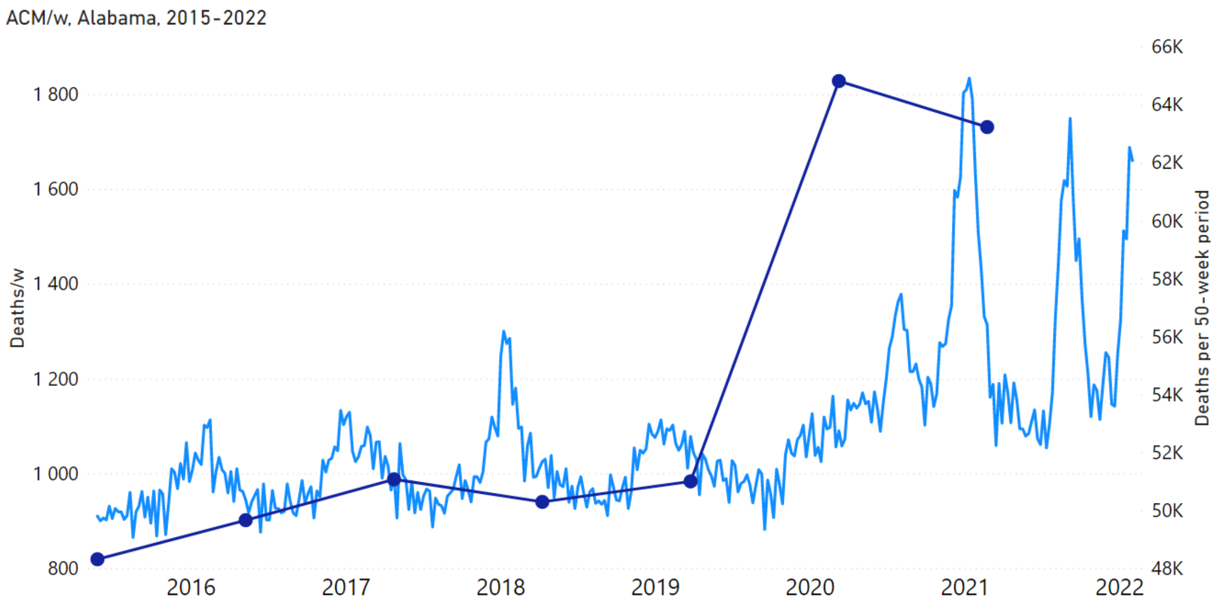
Appendix

The Appendix is in three parts:

- **Appendix A** shows for each state of the USA:
  - ACM/w versus time together with ACM by 50-week period, from 2015 to 2022 (equivalent to Figure 12 without the color-coded periods)
  - Excess mortality of the pre-vaccination and vaccination periods of the covid period, by age group (equivalent to Table 4Table 5)
  - Excess mortality of the covid period, by age group (equivalent to Table 3)
- **Appendix B** shows state-wise maps of poverty and obesity in the USA
- **Appendix C** shows ACM/w in the USA with most recent data, from 2015 to 2022

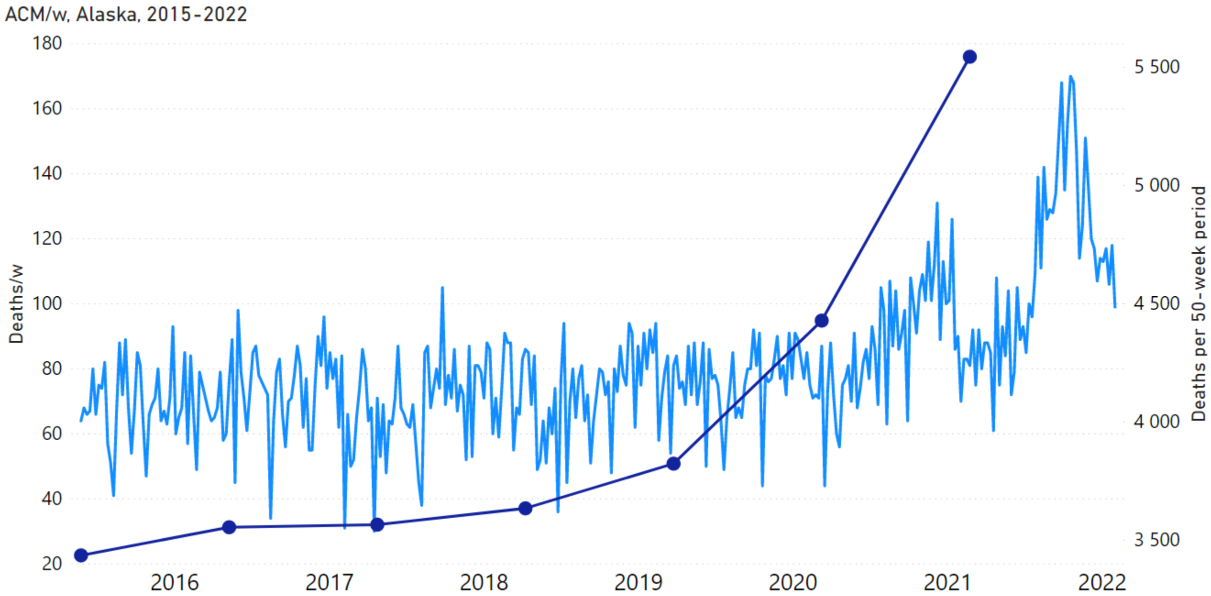
The states in Appendix A are ordered alphabetically.

Appendix A – ACM/w and by 50-week period, by state, 2015-2022



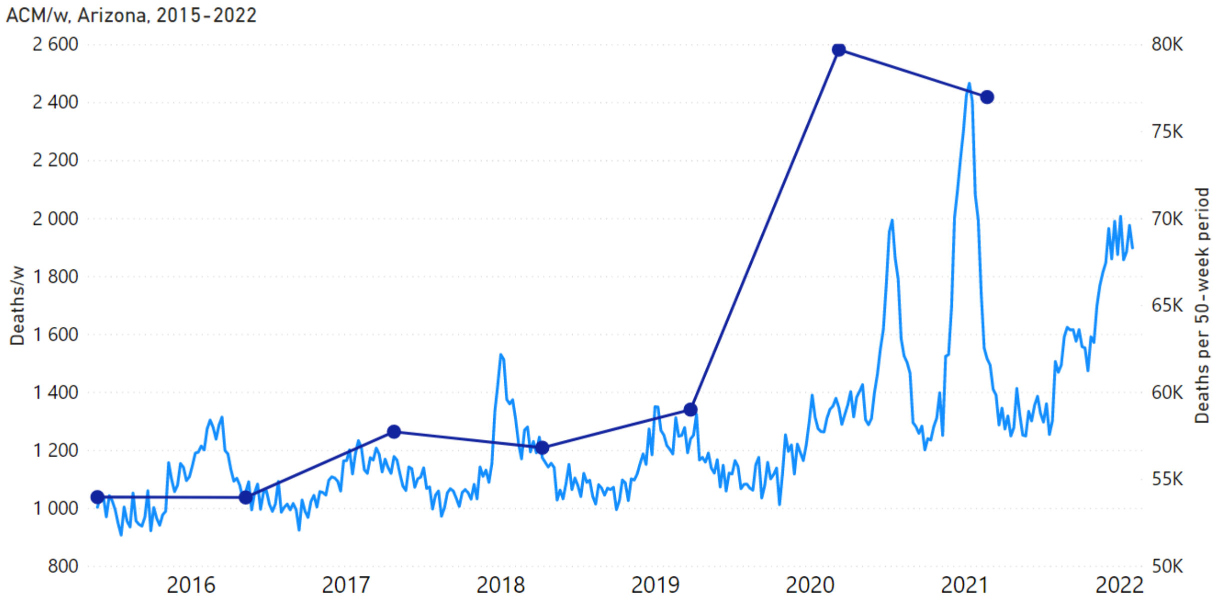
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Alabama**	**63 285**	**64 837**	**51 016**	**13 821**	**12 269**	**27,09 %**	**24,05 %**
0-24	1 406	1 278	1 257	21	149	1,67 %	11,85 %
25-44	4 124	3 569	2 785	784	1 339	28,15 %	48,08 %
45-64	14 580	13 643	10 908	2 735	3 672	25,07 %	33,66 %
65-74	14 557	14 404	11 148	3 256	3 409	29,21 %	30,58 %
75-84	15 247	16 541	12 570	3 971	2 677	31,59 %	21,30 %
85+	13 371	15 402	12 348	3 054	1 023	24,73 %	8,28 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Alabama**	**128 122**	**101 323**	**100 764**	**26 799**	**27 358**	**26,45 %**	**27,15 %**
0-24	2 684	2 399	2 599	285	85	11,88 %	3,27 %
25-44	7 693	5 501	5 643	2 192	2 050	39,85 %	36,33 %
45-64	28 223	21 520	22 021	6 703	6 202	31,15 %	28,16 %
65-74	28 961	21 897	21 240	7 064	7 721	32,26 %	36,35 %
75-84	31 788	25 293	24 520	6 495	7 268	25,68 %	29,64 %
85+	28 773	24 713	24 741	4 060	4 032	16,43 %	16,30 %



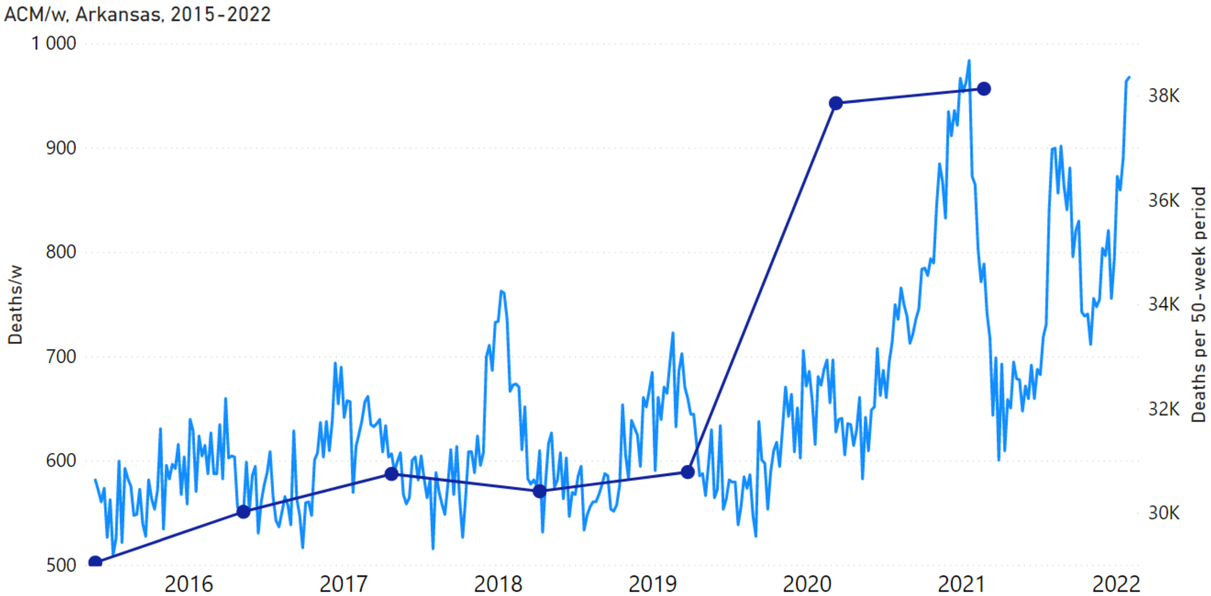
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Alaska**	**5 574**	**4 429**	**3 824**	**605**	**1 750**	**15,82 %**	**45,76 %**
0-24			11	-11	-11	-100,00 %	-100,00 %
25-44	553	317	174	143	379	82,18 %	217,82 %
45-64	1 477	1 201	1 039	162	438	15,59 %	42,16 %
65-74	1 378	1 035	922	113	456	12,26 %	49,46 %
75-84	1 215	1 041	897	144	318	16,05 %	35,45 %
85+	951	835	781	54	170	6,91 %	21,77 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Alaska**	**10 003**	**7 459**	**7 121**	**2 544**	**2 882**	**34,11 %**	**40,47 %**
0-24		11	11	-11	-11	-100,00 %	-100,00 %
25-44	870	300	324	570	546	190,00 %	168,52 %
45-64	2 678	2 104	2 208	574	470	27,28 %	21,29 %
65-74	2 413	1 820	1 687	593	726	32,58 %	43,03 %
75-84	2 256	1 767	1 510	489	746	27,67 %	49,40 %
85+	1 786	1 457	1 381	329	405	22,58 %	29,33 %



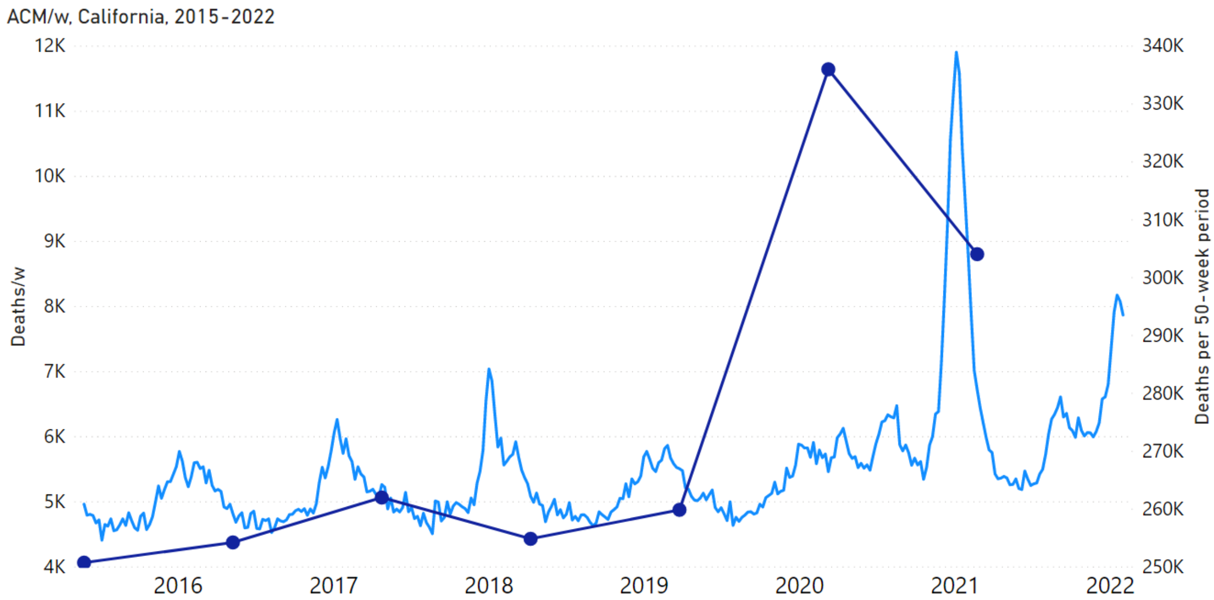
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Arizona**	**77 054**	**79 720**	**59 017**	**20 703**	**18 037**	**35,08 %**	**30,56 %**
0-24	1 824	1 680	1 481	199	343	13,44 %	23,16 %
25-44	5 474	4 901	3 437	1 464	2 037	42,60 %	59,27 %
45-64	15 096	15 080	10 615	4 465	4 481	42,06 %	42,21 %
65-74	16 313	16 605	11 803	4 802	4 510	40,68 %	38,21 %
75-84	19 514	20 528	15 045	5 483	4 469	36,44 %	29,70 %
85+	18 833	20 926	16 636	4 290	2 197	25,79 %	13,21 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Arizona**	**156 774**	**115 842**	**111 708**	**40 932**	**45 066**	**35,33 %**	**40,34 %**
0-24	3 504	3 005	2 801	499	703	16,61 %	25,10 %
25-44	10 375	6 590	6 040	3 785	4 335	57,44 %	71,77 %
45-64	30 176	21 229	20 940	8 947	9 236	42,15 %	44,11 %
65-74	32 918	22 977	21 975	9 941	10 943	43,27 %	49,80 %
75-84	40 042	29 379	27 485	10 663	12 557	36,29 %	45,69 %
85+	39 759	32 662	32 467	7 097	7 292	21,73 %	22,46 %



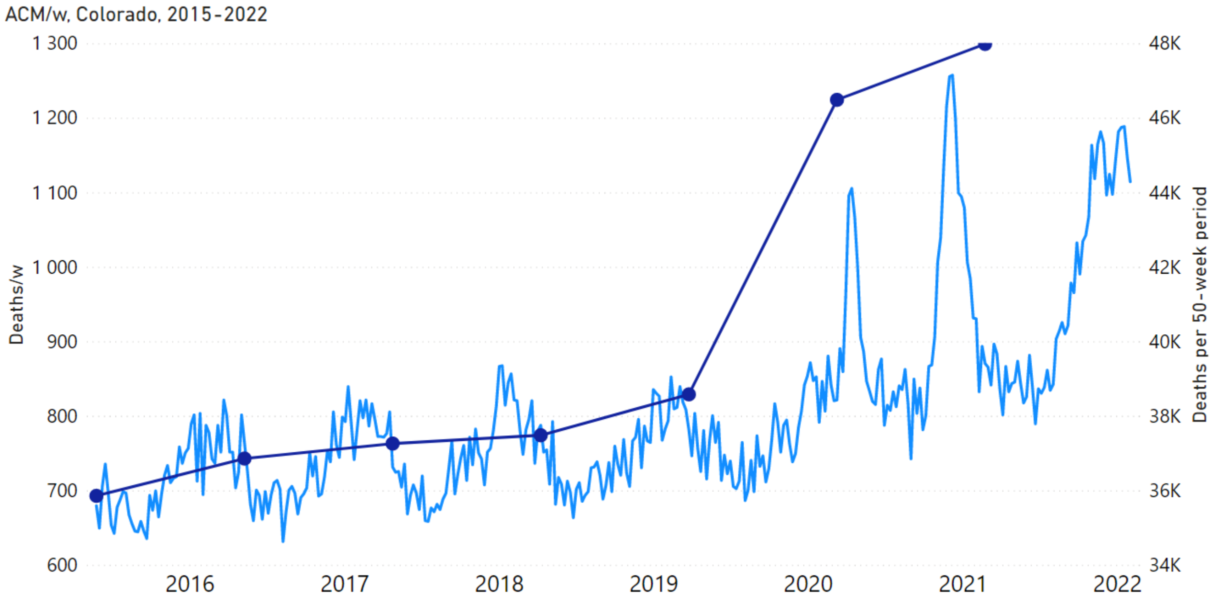
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Arkansas**	**38 165**	**37 863**	**30 791**	**7 072**	**7 374**	**22,97 %**	**23,95 %**
0-24	821	721	667	54	154	8,10 %	23,09 %
25-44	2 393	1 974	1 518	456	875	30,04 %	57,64 %
45-64	8 490	7 772	6 232	1 540	2 258	24,71 %	36,23 %
65-74	8 675	8 322	6 632	1 690	2 043	25,48 %	30,81 %
75-84	9 339	9 566	7 871	1 695	1 468	21,53 %	18,65 %
85+	8 447	9 508	7 871	1 637	576	20,80 %	7,32 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Arkansas**	**76 028**	**61 214**	**60 784**	**14 814**	**15 244**	**24,20 %**	**25,08 %**
0-24	1 542	1 239	1 291	303	251	24,46 %	19,44 %
25-44	4 367	3 026	3 021	1 341	1 346	44,32 %	44,55 %
45-64	16 262	12 616	12 855	3 646	3 407	28,90 %	26,50 %
65-74	16 997	13 042	12 807	3 955	4 190	30,33 %	32,72 %
75-84	18 905	15 754	15 037	3 151	3 868	20,00 %	25,72 %
85+	17 955	15 537	15 773	2 418	2 182	15,56 %	13,83 %



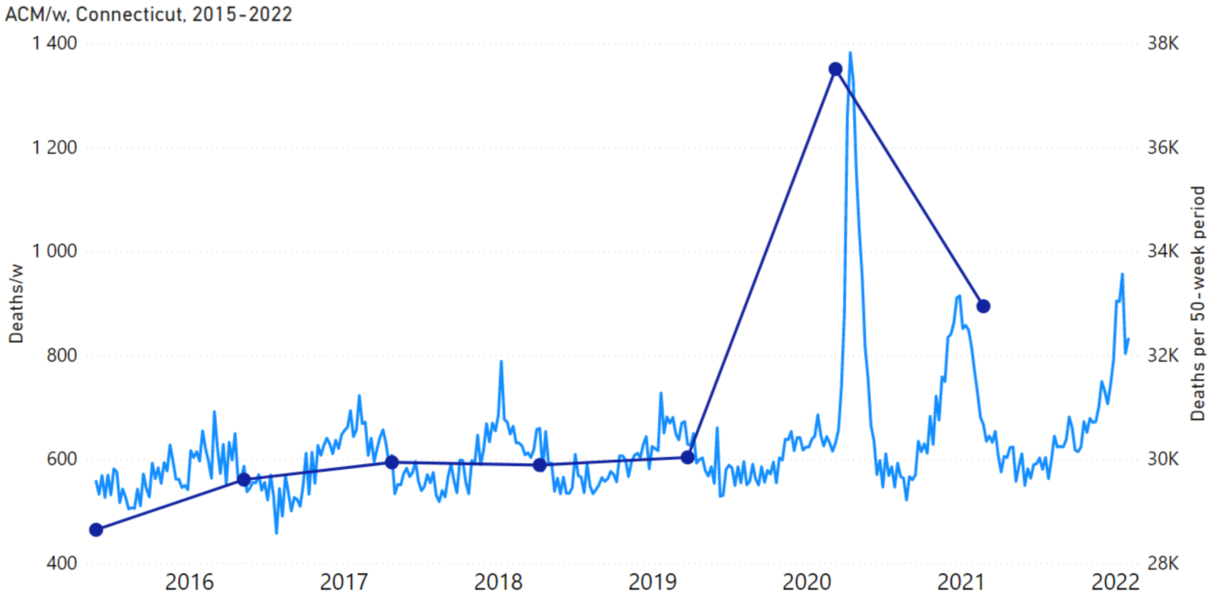
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **California**	**304 271**	**335 996**	**259 882**	**76 114**	**44 389**	**29,29 %**	**17,08 %**
0-24	6 141	6 272	5 470	802	671	14,66 %	12,27 %
25-44	19 971	18 694	13 342	5 352	6 629	40,11 %	49,69 %
45-64	59 819	64 752	47 587	17 165	12 232	36,07 %	25,70 %
65-74	58 165	64 885	47 750	17 135	10 415	35,88 %	21,81 %
75-84	68 779	77 266	60 120	17 146	8 659	28,52 %	14,40 %
85+	91 396	104 127	85 613	18 514	5 783	21,63 %	6,75 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **California**	**640 267**	**514 752**	**516 253**	**125 515**	**124 014**	**24,38 %**	**24,02 %**
0-24	12 413	10 837	11 720	1 576	693	14,54 %	5,91 %
25-44	38 665	25 834	24 117	12 831	14 548	49,67 %	60,32 %
45-64	124 571	95 294	96 943	29 277	27 628	30,72 %	28,50 %
65-74	123 050	94 336	91 863	28 714	31 187	30,44 %	33,95 %
75-84	146 045	118 457	116 313	27 588	29 732	23,29 %	25,56 %
85+	195 523	169 994	175 297	25 529	20 226	15,02 %	11,54 %



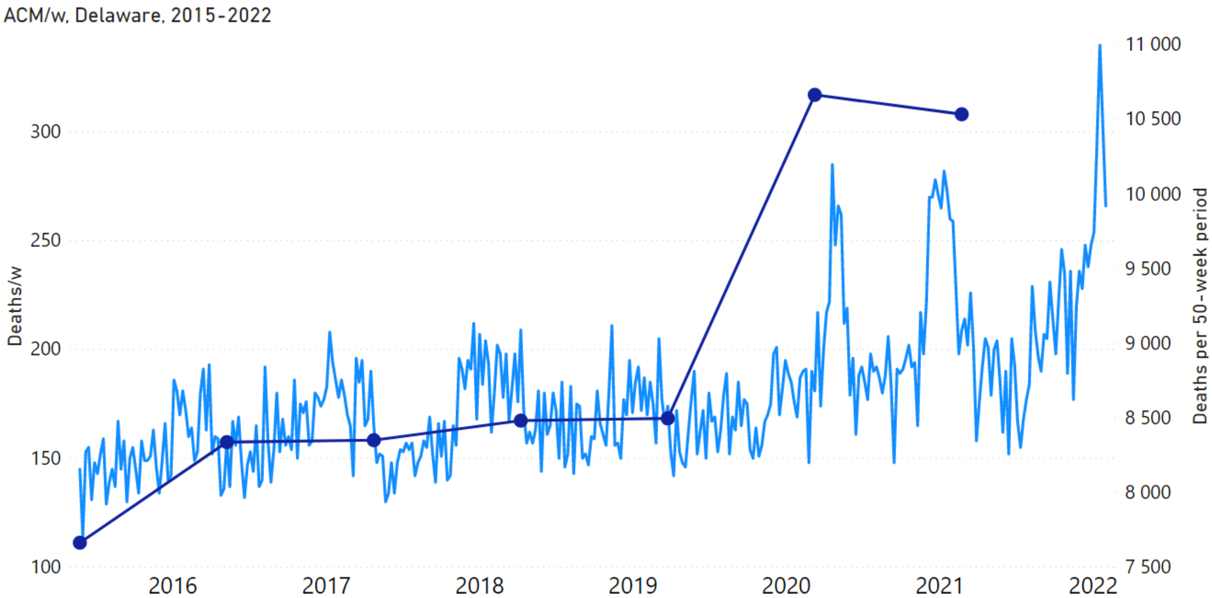
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Colorado	**48 081**	**46 498**	**38 591**	**7 907**	**9 490**	**20,49 %**	**24,59 %**
0-24	1 210	1 159	1 045	114	165	10,91 %	15,79 %
25-44	3 775	3 175	2 516	659	1 259	26,19 %	50,04 %
45-64	9 580	8 505	7 265	1 240	2 315	17,07 %	31,87 %
65-74	9 768	9 003	7 362	1 641	2 406	22,29 %	32,68 %
75-84	11 130	10 861	8 820	2 041	2 310	23,14 %	26,19 %
85+	12 618	13 795	11 583	2 212	1 035	19,10 %	8,94 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Colorado	**94 579**	**76 084**	**74 138**	**18 495**	**20 441**	**24,31 %**	**27,57 %**
0-24	2 369	2 064	2 091	305	278	14,78 %	13,30 %
25-44	6 950	4 895	4 486	2 055	2 464	41,98 %	54,93 %
45-64	18 085	14 573	14 595	3 512	3 490	24,10 %	23,91 %
65-74	18 771	14 370	13 396	4 401	5 375	30,63 %	40,12 %
75-84	21 991	17 284	16 518	4 707	5 473	27,23 %	33,13 %
85+	26 413	22 898	23 052	3 515	3 361	15,35 %	14,58 %



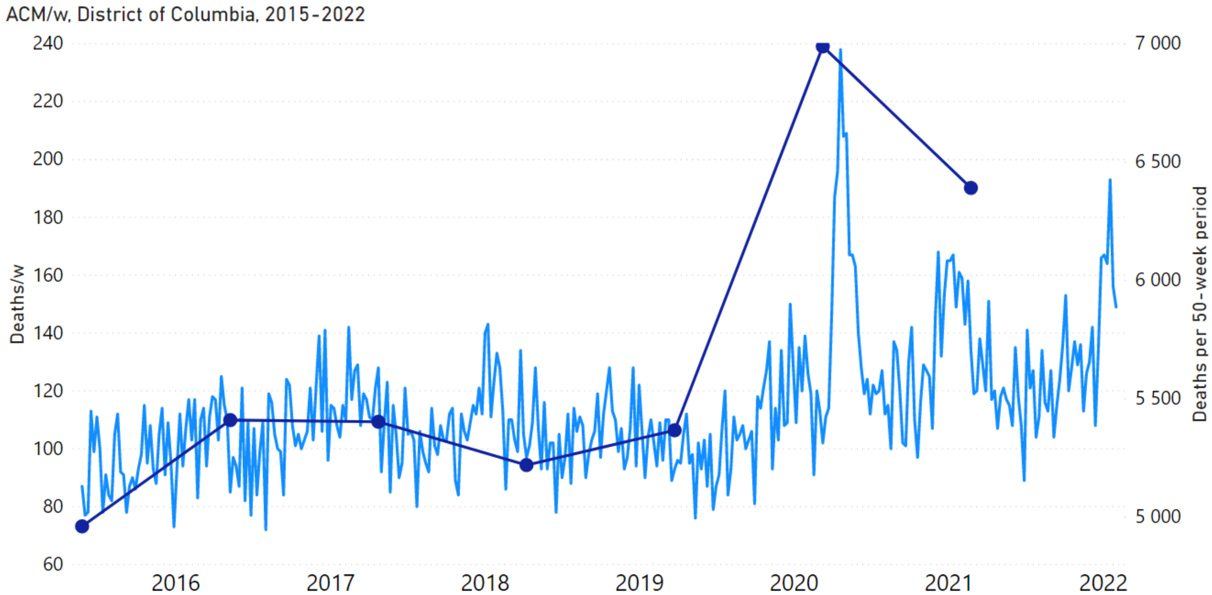
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Connecticut**	**32 992**	**37 514**	**30 041**	**7 473**	**2 951**	**24,88 %**	**9,82 %**
0-24	249	178	179	-1	70	-0,56 %	39,11 %
25-44	1 692	1 604	1 369	235	323	17,17 %	23,59 %
45-64	5 812	6 074	4 825	1 249	987	25,89 %	20,46 %
65-74	5 781	6 268	4 984	1 284	797	25,76 %	15,99 %
75-84	7 893	8 926	6 913	2 013	980	29,12 %	14,18 %
85+	11 565	14 464	11 771	2 693	-206	22,88 %	-1,75 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Connecticut**	**70 506**	**59 934**	**59 556**	**10 572**	**10 950**	**17,64 %**	**18,39 %**
0-24	427	360	404	67	23	18,61 %	5,69 %
25-44	3 296	2 629	2 430	667	866	25,37 %	35,64 %
45-64	11 886	9 675	9 517	2 211	2 369	22,85 %	24,89 %
65-74	12 049	9 768	9 596	2 281	2 453	23,35 %	25,56 %
75-84	16 819	13 833	13 254	2 986	3 565	21,59 %	26,90 %
85+	26 029	23 669	24 355	2 360	1 674	9,97 %	6,87 %



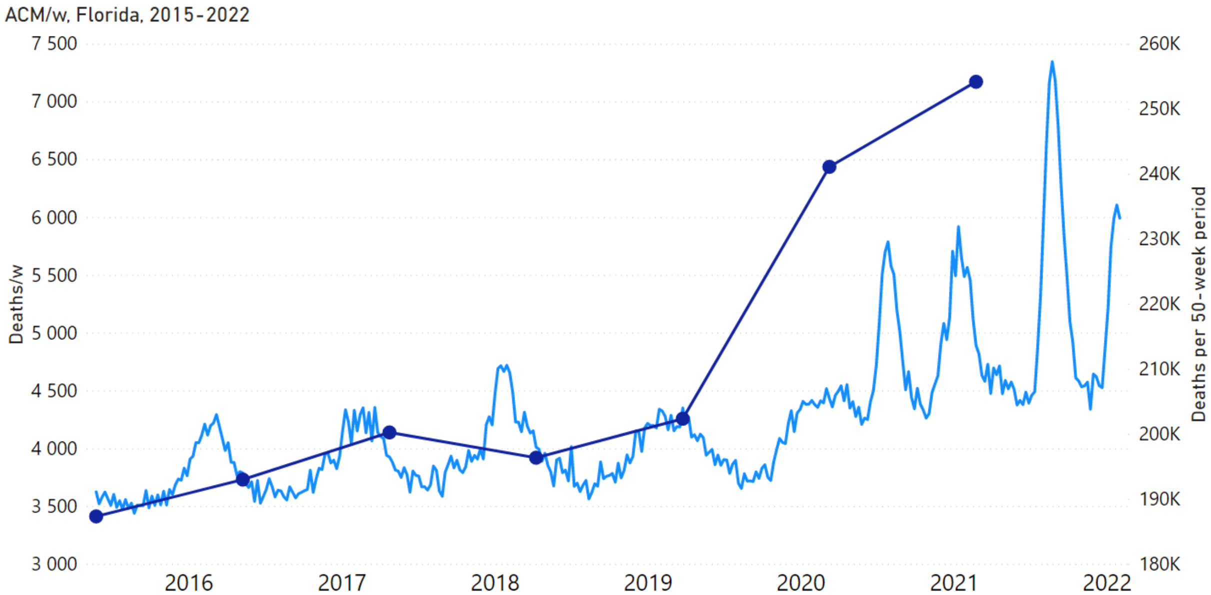
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Delaware	**10 568**	**10 665**	**8 497**	**2 168**	**2 071**	**25,51 %**	**24,37 %**
25-44	468	340	344	-4	124	-1,16 %	36,05 %
45-64	2 080	1 927	1 627	300	453	18,44 %	27,84 %
65-74	2 364	2 271	1 721	550	643	31,96 %	37,36 %
75-84	2 750	2 849	2 173	676	577	31,11 %	26,55 %
85+	2 906	3 278	2 632	646	274	24,54 %	10,41 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Delaware	**21 233**	**16 979**	**16 689**	**4 254**	**4 544**	**25,05 %**	**27,23 %**
0-24			23		-23		-100,00 %
25-44	808	573	342	235	466	41,01 %	136,26 %
45-64	4 007	3 224	3 399	783	608	24,29 %	17,89 %
65-74	4 635	3 433	3 485	1 202	1 150	35,01 %	33,00 %
75-84	5 599	4 387	4 211	1 212	1 388	27,63 %	32,96 %
85+	6 184	5 362	5 229	822	955	15,33 %	18,26 %



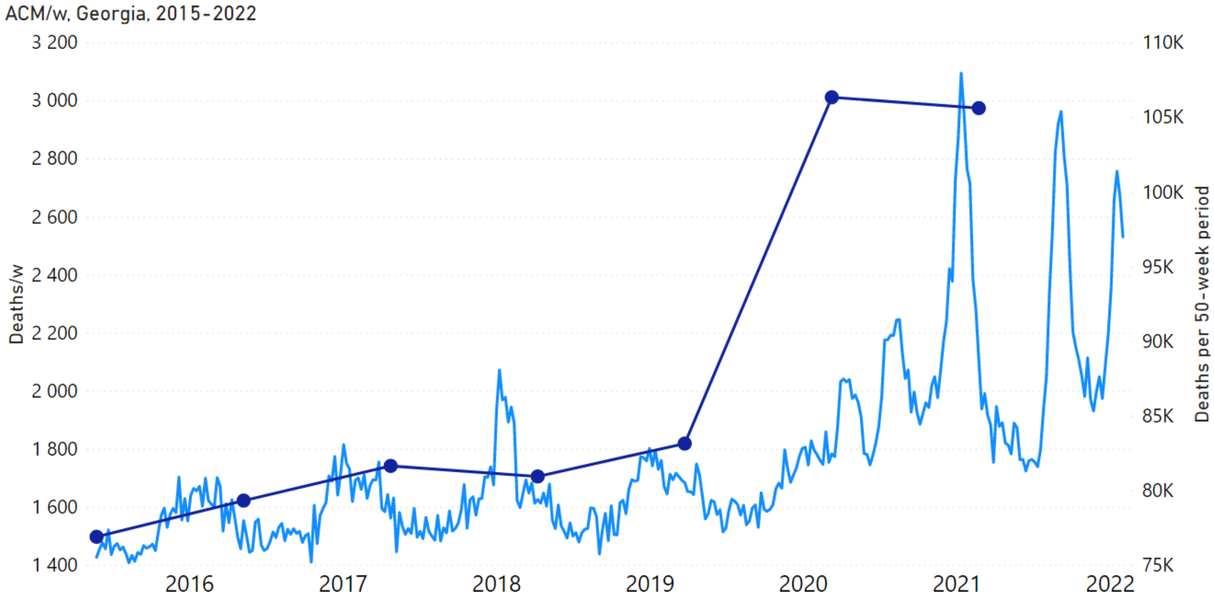
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
District of Columbia	**6 408**	**6 989**	**5 367**	**1 622**	**1 041**	**30,22 %**	**19,40 %**
0-24	11	11	24	-13	-13	-54,17 %	-54,17 %
25-44	538	398	187	211	351	112,83 %	187,70 %
45-64	1 826	2 017	1 596	421	230	26,38 %	14,41 %
65-74	1 549	1 711	1 285	426	264	33,15 %	20,54 %
75-84	1 266	1 377	1 095	282	171	25,75 %	15,62 %
85+	1 218	1 475	1 180	295	38	25,00 %	3,22 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
District of Columbia	**13 397**	**10 587**	**10 813**	**2 810**	**2 584**	**26,54 %**	**23,90 %**
0-24	22	36	59	-14	-37	-38,89 %	-62,71 %
25-44	936	347	291	589	645	169,74 %	221,65 %
45-64	3 843	3 175	3 319	668	524	21,04 %	15,79 %
65-74	3 260	2 511	2 509	749	751	29,83 %	29,93 %
75-84	2 643	2 141	2 145	502	498	23,45 %	23,22 %
85+	2 693	2 377	2 490	316	203	13,29 %	8,15 %



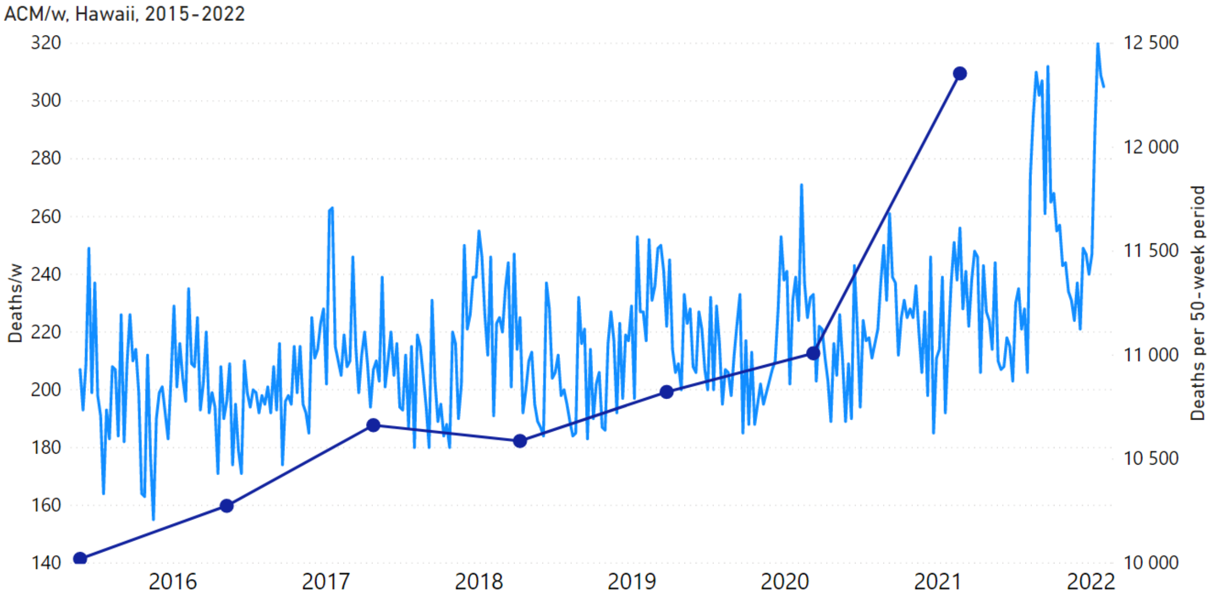
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Florida**	**254 311**	**241 142**	**202 396**	**38 746**	**51 915**	**19,14 %**	**25,65 %**
0-24	4 246	3 914	3 735	179	511	4,79 %	13,68 %
25-44	14 563	12 158	9 761	2 397	4 802	24,56 %	49,20 %
45-64	48 930	42 173	36 005	6 168	12 925	17,13 %	35,90 %
65-74	51 262	47 070	38 774	8 296	12 488	21,40 %	32,21 %
75-84	63 449	61 704	50 625	11 079	12 824	21,88 %	25,33 %
85+	71 861	74 123	63 496	10 627	8 365	16,74 %	13,17 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Florida**	**495 453**	**398 793**	**393 327**	**96 660**	**102 126**	**24,24 %**	**25,96 %**
0-24	8 160	7 465	7 756	695	404	9,31 %	5,21 %
25-44	26 721	18 935	18 746	7 786	7 975	41,12 %	42,54 %
45-64	91 103	72 034	72 573	19 069	18 530	26,47 %	25,53 %
65-74	98 332	76 374	73 622	21 958	24 710	28,75 %	33,56 %
75-84	125 153	99 095	94 775	26 058	30 378	26,30 %	32,05 %
85+	145 984	124 890	125 855	21 094	20 129	16,89 %	15,99 %



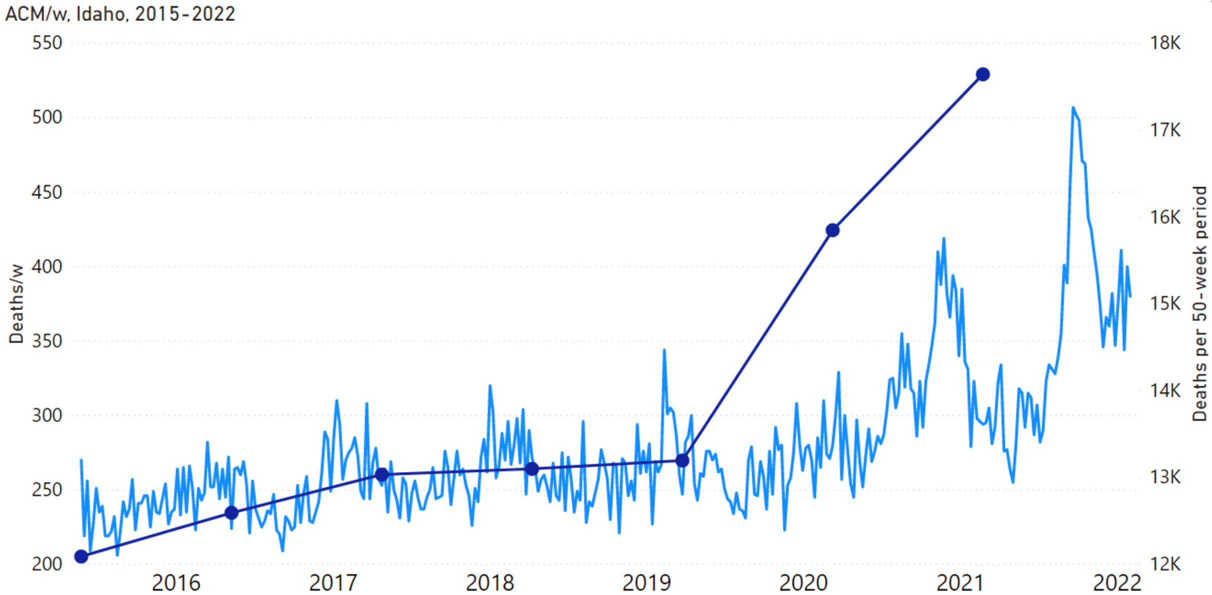
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Georgia**	**105 894**	**106 369**	**83 159**	**23 210**	**22 735**	**27,91 %**	**27,34 %**
0-24	2 536	2 336	2 241	95	295	4,24 %	13,16 %
25-44	7 315	6 344	4 711	1 633	2 604	34,66 %	55,27 %
45-64	24 759	23 038	18 416	4 622	6 343	25,10 %	34,44 %
65-74	23 743	23 429	17 926	5 503	5 817	30,70 %	32,45 %
75-84	25 142	26 114	19 957	6 157	5 185	30,85 %	25,98 %
85+	22 399	25 108	19 908	5 200	2 491	26,12 %	12,51 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Georgia**	**212 263**	**164 114**	**161 001**	**48 149**	**51 262**	**29,34 %**	**31,84 %**
0-24	4 872	4 485	4 718	387	154	8,63 %	3,26 %
25-44	13 659	9 269	9 126	4 390	4 533	47,36 %	49,67 %
45-64	47 797	36 462	36 733	11 335	11 064	31,09 %	30,12 %
65-74	47 172	35 484	33 934	11 688	13 238	32,94 %	39,01 %
75-84	51 256	38 993	37 373	12 263	13 883	31,45 %	37,15 %
85+	47 507	39 421	39 117	8 086	8 390	20,51 %	21,45 %



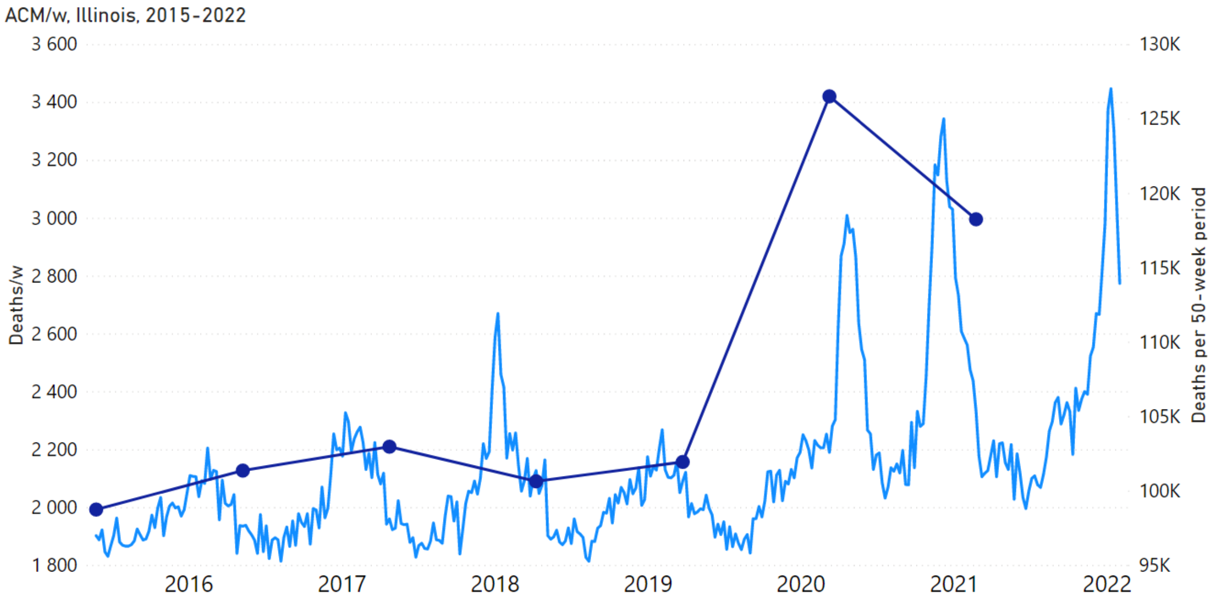
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Hawaii	**12 364**	**11 009**	**10 823**	**186**	**1 541**	**1,72 %**	**14,24 %**
25-44	439	260	285	-25	154	-8,77 %	54,04 %
45-64	2 300	2 002	1 955	47	345	2,40 %	17,65 %
65-74	2 509	2 256	2 068	188	441	9,09 %	21,32 %
75-84	2 747	2 448	2 299	149	448	6,48 %	19,49 %
85+	4 369	4 043	4 216	-173	153	-4,10 %	3,63 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Hawaii	**23 373**	**21 410**	**20 938**	**1 963**	**2 435**	**9,17 %**	**11,63 %**
0-24		13	11	-13	-11	-100,00 %	-100,00 %
25-44	699	505	386	194	313	38,42 %	81,09 %
45-64	4 302	3 901	3 888	401	414	10,28 %	10,65 %
65-74	4 765	4 173	3 986	592	779	14,19 %	19,54 %
75-84	5 195	4 568	4 512	627	683	13,73 %	15,14 %
85+	8 412	8 250	8 155	162	257	1,96 %	3,15 %



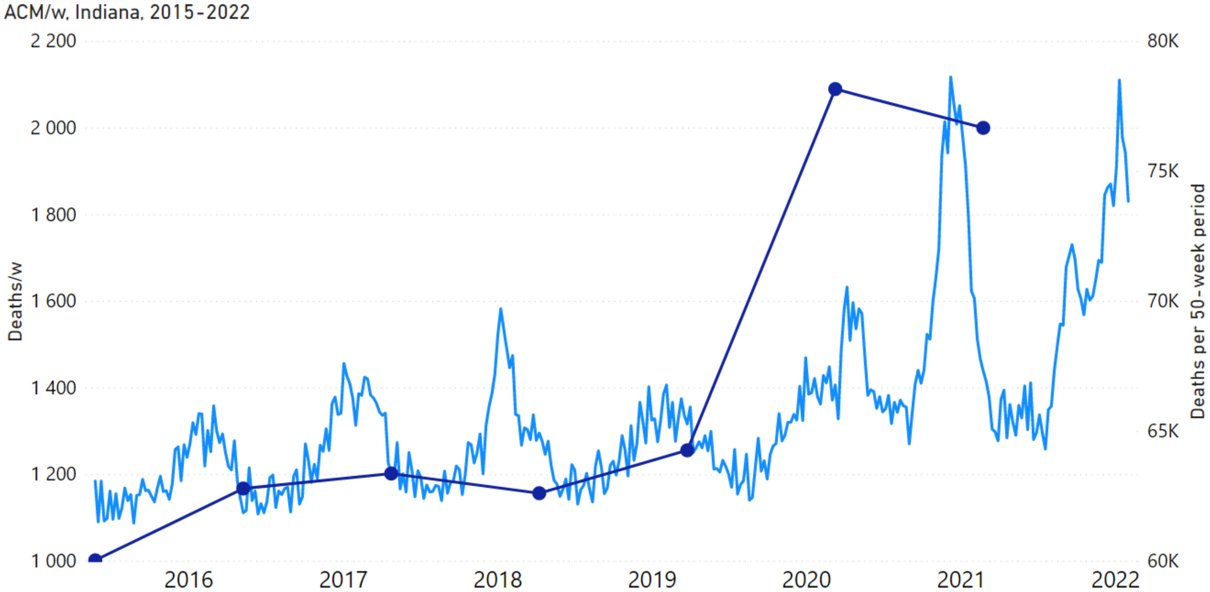
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Idaho	**17 644**	**15 848**	**13 195**	**2 653**	**4 449**	**20,11 %**	**33,72 %**
0-24	85	92	35	57	50	162,86 %	142,86 %
25-44	885	710	342	368	543	107,60 %	158,77 %
45-64	3 203	2 631	2 216	415	987	18,73 %	44,54 %
65-74	3 838	3 234	2 647	587	1 191	22,18 %	44,99 %
75-84	4 638	4 292	3 670	622	968	16,95 %	26,38 %
85+	4 995	4 889	4 285	604	710	14,10 %	16,57 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Idaho	**33 492**	**26 294**	**25 626**	**7 198**	**7 866**	**27,38 %**	**30,70 %**
0-24	177	76	109	101	68	132,89 %	62,39 %
25-44	1 595	784	861	811	734	103,44 %	85,25 %
45-64	5 834	4 436	4 482	1 398	1 352	31,51 %	30,17 %
65-74	7 072	5 334	5 034	1 738	2 038	32,58 %	40,48 %
75-84	8 930	7 136	6 777	1 794	2 153	25,14 %	31,77 %
85+	9 884	8 528	8 363	1 356	1 521	15,90 %	18,19 %



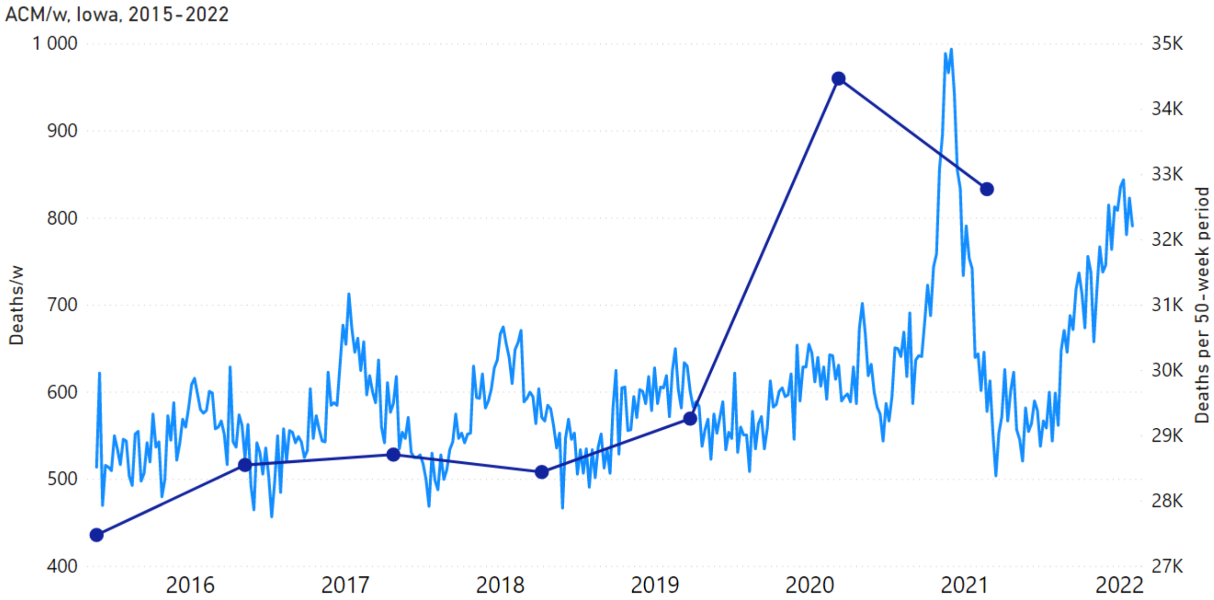
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Illinois	**118 278**	**126 531**	**101 962**	**24 569**	**16 316**	**24,10 %**	**16,00 %**
0-24	2 342	2 264	1 999	265	343	13,26 %	17,16 %
25-44	6 561	6 078	4 869	1 209	1 692	24,83 %	34,75 %
45-64	23 031	22 956	18 692	4 264	4 339	22,81 %	23,21 %
65-74	24 066	24 816	19 398	5 418	4 668	27,93 %	24,06 %
75-84	27 993	30 258	24 060	6 198	3 933	25,76 %	16,35 %
85+	34 285	40 159	32 944	7 215	1 341	21,90 %	4,07 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Illinois	**244 809**	**202 621**	**204 365**	**42 188**	**40 444**	**20,82 %**	**19,79 %**
0-24	4 606	4 153	4 871	453	-265	10,91 %	-5,44 %
25-44	12 639	9 618	9 432	3 021	3 207	31,41 %	34,00 %
45-64	45 987	37 349	37 681	8 638	8 306	23,13 %	22,04 %
65-74	48 882	37 890	36 761	10 992	12 121	29,01 %	32,97 %
75-84	58 251	47 770	47 456	10 481	10 795	21,94 %	22,75 %
85+	74 444	65 841	68 164	8 603	6 280	13,07 %	9,21 %



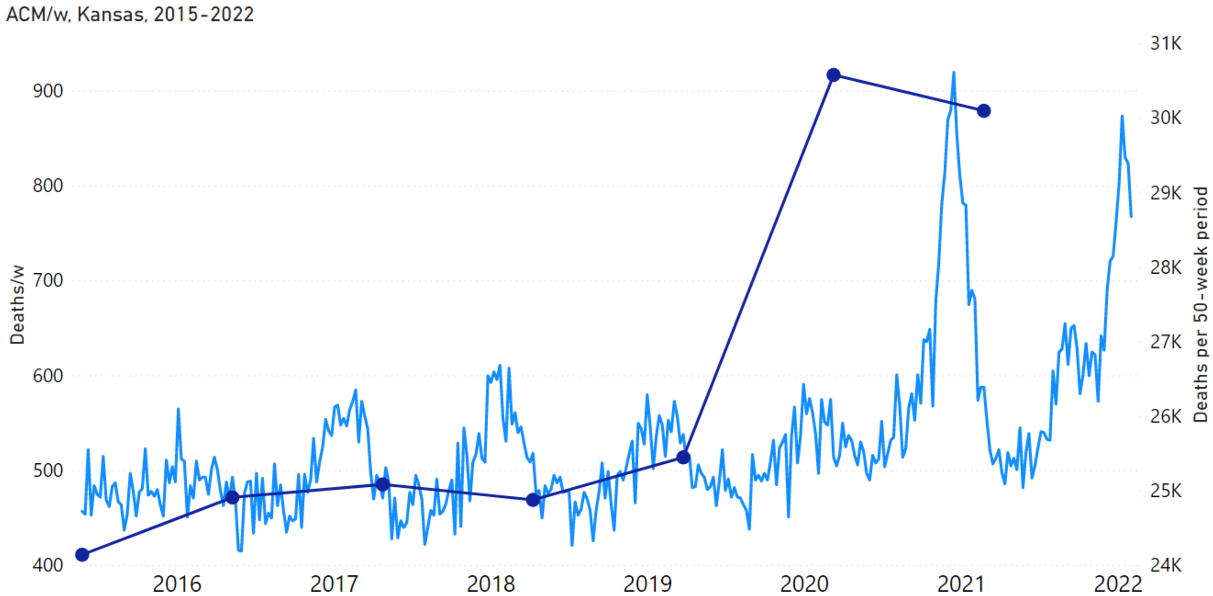
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Indiana**	**76 829**	**78 173**	**64 270**	**13 903**	**12 559**	**21,63 %**	**19,54 %**
0-24	1 665	1 604	1 416	188	249	13,28 %	17,58 %
25-44	4 644	4 151	3 271	880	1 373	26,90 %	41,97 %
45-64	15 699	14 535	12 203	2 332	3 496	19,11 %	28,65 %
65-74	16 899	15 906	13 091	2 815	3 808	21,50 %	29,09 %
75-84	18 571	19 361	15 660	3 701	2 911	23,63 %	18,59 %
85+	19 351	22 616	18 629	3 987	722	21,40 %	3,88 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Indiana**	**155 002**	**126 889**	**126 175**	**28 113**	**28 827**	**22,16 %**	**22,85 %**
0-24	3 269	2 867	3 072	402	197	14,02 %	6,41 %
25-44	8 795	6 374	6 500	2 421	2 295	37,98 %	35,31 %
45-64	30 234	24 538	24 912	5 696	5 322	23,21 %	21,36 %
65-74	32 805	25 463	23 769	7 342	9 036	28,83 %	38,02 %
75-84	37 932	30 788	30 109	7 144	7 823	23,20 %	25,98 %
85+	41 967	36 859	37 813	5 108	4 154	13,86 %	10,99 %



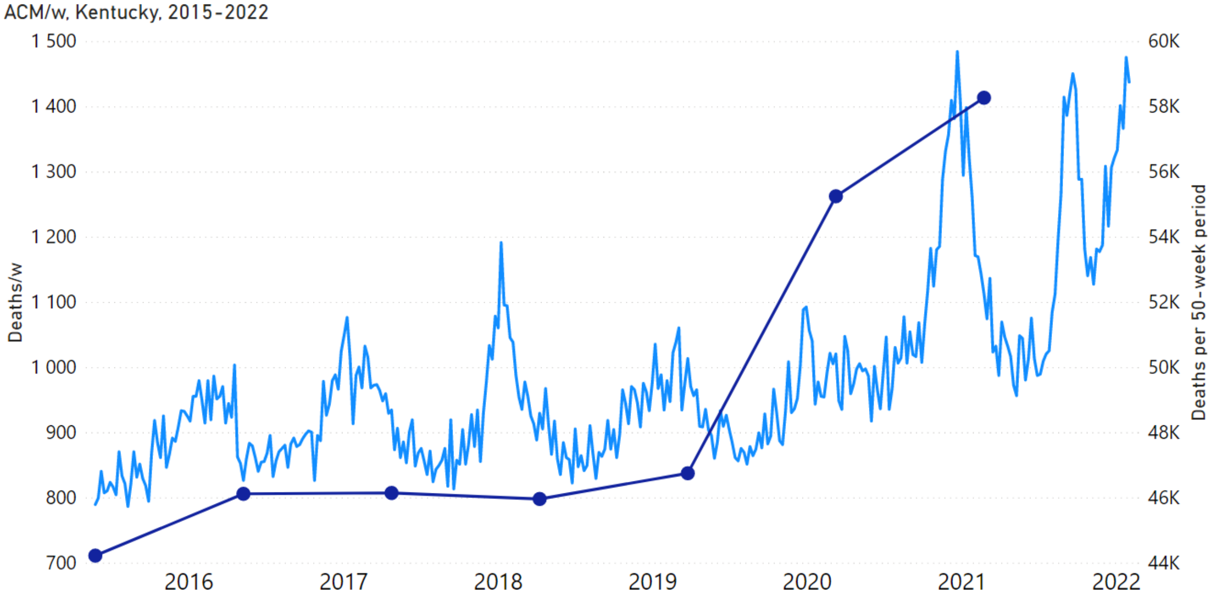
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Iowa**	**32 816**	**34 471**	**29 263**	**5 208**	**3 553**	**17,80 %**	**12,14 %**
0-24	158	286	241	45	-83	18,67 %	-34,44 %
25-44	1 331	1 264	1 034	230	297	22,24 %	28,72 %
45-64	5 695	5 394	4 761	633	934	13,30 %	19,62 %
65-74	6 725	6 485	5 255	1 230	1 470	23,41 %	27,97 %
75-84	8 171	8 417	7 205	1 212	966	16,82 %	13,41 %
85+	10 736	12 625	10 767	1 858	-31	17,26 %	-0,29 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Iowa**	**67 287**	**57 708**	**57 265**	**9 579**	**10 022**	**16,60 %**	**17,50 %**
0-24	444	461	689	-17	-245	-3,69 %	-35,56 %
25-44	2 595	1 979	1 871	616	724	31,13 %	38,70 %
45-64	11 089	9 371	9 268	1 718	1 821	18,33 %	19,65 %
65-74	13 210	10 376	9 819	2 834	3 391	27,31 %	34,54 %
75-84	16 588	14 159	13 598	2 429	2 990	17,16 %	21,99 %
85+	23 361	21 362	22 020	1 999	1 341	9,36 %	6,09 %



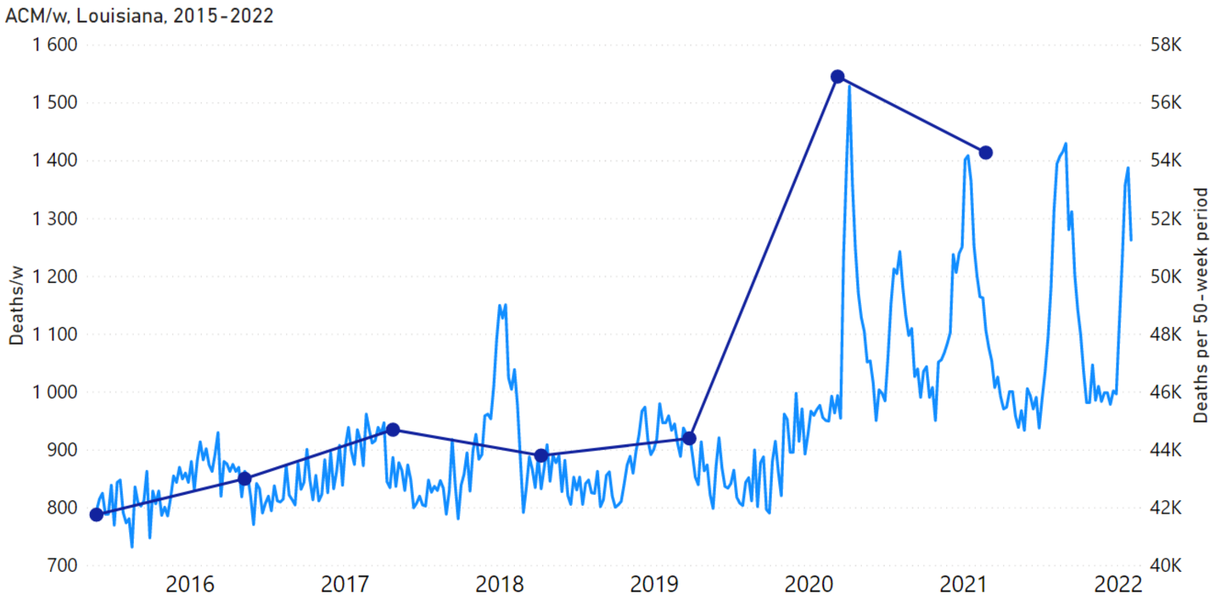
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Kansas**	**30 110**	**30 583**	**25 450**	**5 133**	**4 660**	**20,17 %**	**18,31 %**
0-24	458	409	356	53	102	14,89 %	28,65 %
25-44	1 653	1 410	1 133	277	520	24,45 %	45,90 %
45-64	5 639	5 243	4 497	746	1 142	16,59 %	25,39 %
65-74	6 289	5 949	4 848	1 101	1 441	22,71 %	29,72 %
75-84	7 217	7 452	6 080	1 372	1 137	22,57 %	18,70 %
85+	8 854	10 120	8 536	1 584	318	18,56 %	3,73 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Kansas**	**60 693**	**50 329**	**49 999**	**10 364**	**10 694**	**20,59 %**	**21,39 %**
0-24	867	644	813	223	54	34,63 %	6,64 %
25-44	3 063	2 203	2 072	860	991	39,04 %	47,83 %
45-64	10 882	8 826	9 066	2 056	1 816	23,29 %	20,03 %
65-74	12 238	9 565	8 879	2 673	3 359	27,95 %	37,83 %
75-84	14 669	11 964	11 924	2 705	2 745	22,61 %	23,02 %
85+	18 974	17 127	17 245	1 847	1 729	10,78 %	10,03 %



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Kentucky**	**58 357**	**55 261**	**46 760**	**8 501**	**11 597**	**18,18 %**	**24,80 %**
0-24	1 068	1 034	771	263	297	34,11 %	38,52 %
25-44	3 891	3 546	2 548	998	1 343	39,17 %	52,71 %
45-64	13 668	11 974	10 343	1 631	3 325	15,77 %	32,15 %
65-74	13 413	11 993	10 034	1 959	3 379	19,52 %	33,68 %
75-84	14 165	13 426	11 649	1 777	2 516	15,25 %	21,60 %
85+	12 152	13 288	11 415	1 873	737	16,41 %	6,46 %

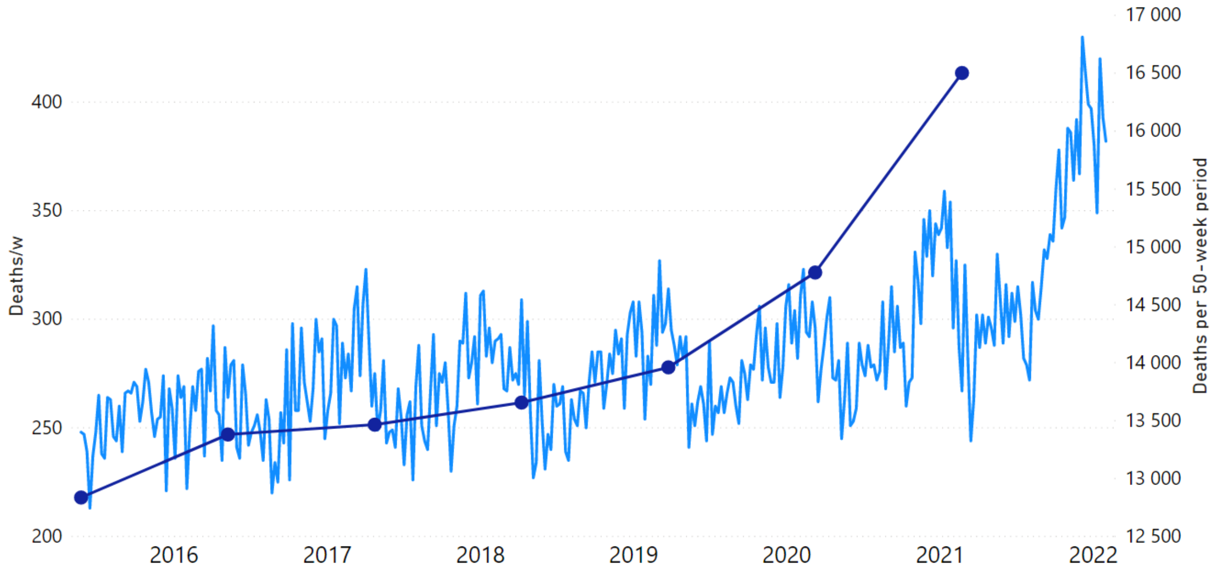
State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Kentucky**	**113 618**	**92 731**	**92 284**	**20 887**	**21 334**	**22,52 %**	**23,12 %**
0-24	2 102	1 549	1 801	553	301	35,70 %	16,71 %
25-44	7 437	5 136	5 419	2 301	2 018	44,80 %	37,24 %
45-64	25 642	20 508	20 939	5 134	4 703	25,03 %	22,46 %
65-74	25 406	19 978	19 322	5 428	6 084	27,17 %	31,49 %
75-84	27 591	22 933	22 005	4 658	5 586	20,31 %	25,39 %
85+	25 440	22 627	22 798	2 813	2 642	12,43 %	11,59 %



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Louisiana	**54 566**	**56 915**	**44 401**	**12 514**	**10 165**	**28,18 %**	**22,89 %**
0-24	1 427	1 363	1 228	135	199	10,99 %	16,21 %
25-44	4 318	3 856	2 853	1 003	1 465	35,16 %	51,35 %
45-64	12 638	12 261	9 786	2 475	2 852	25,29 %	29,14 %
65-74	12 109	12 467	9 390	3 077	2 719	32,77 %	28,96 %
75-84	12 456	13 540	10 437	3 103	2 019	29,73 %	19,34 %
85+	11 618	13 428	10 707	2 721	911	25,41 %	8,51 %

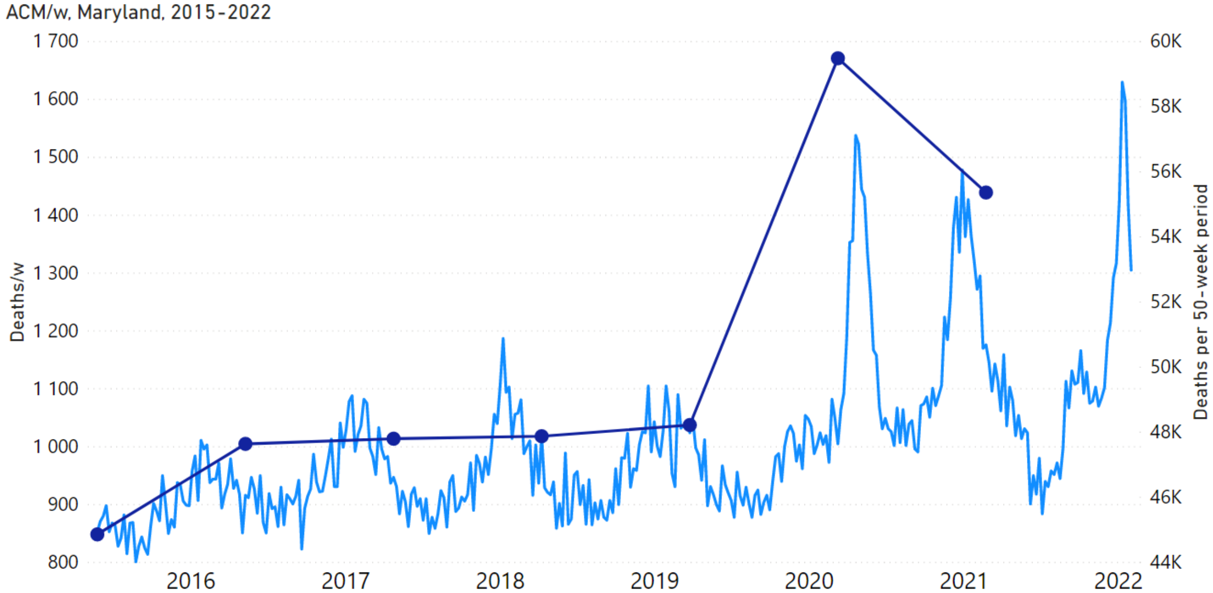
State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Louisiana	**111 481**	**88 207**	**87 706**	**23 274**	**23 775**	**26,39 %**	**27,11 %**
0-24	2 790	2 419	2 553	371	237	15,34 %	9,28 %
25-44	8 174	5 493	5 505	2 681	2 669	48,81 %	48,48 %
45-64	24 899	19 582	20 137	5 317	4 762	27,15 %	23,65 %
65-74	24 576	18 668	17 936	5 908	6 640	31,65 %	37,02 %
75-84	25 996	20 673	20 090	5 323	5 906	25,75 %	29,40 %
85+	25 046	21 372	21 485	3 674	3 561	17,19 %	16,57 %

ACM/w, Maine, 2015-2022



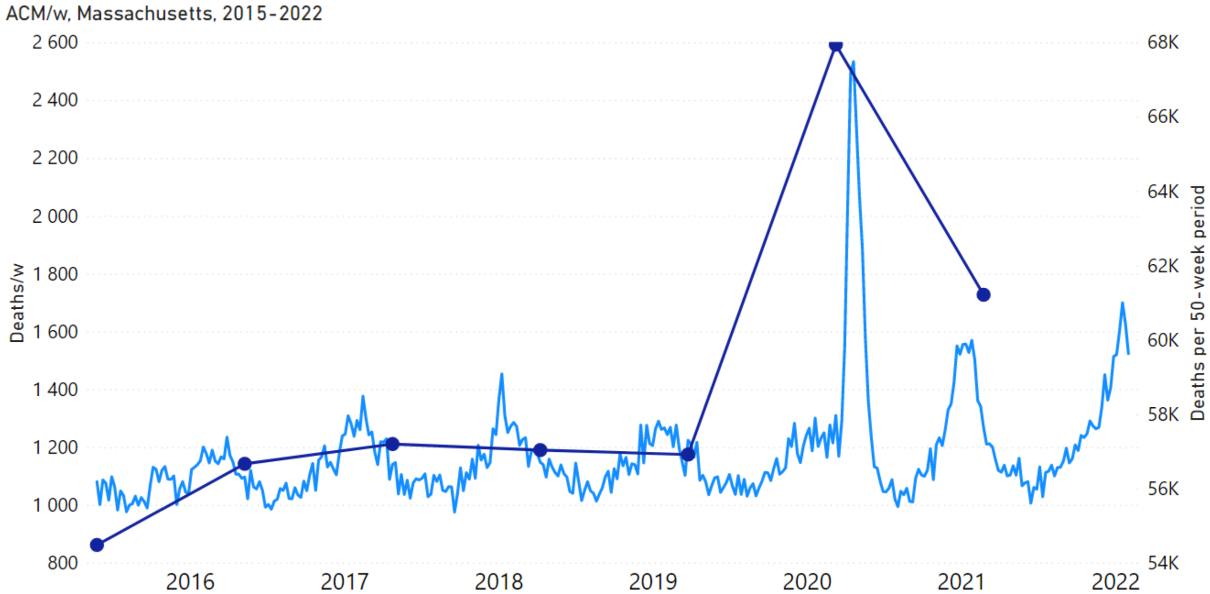
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Maine	**16 503**	**14 779**	**13 960**	**819**	**2 543**	**5,87 %**	**18,22 %**
25-44	769	501	439	62	330	14,12 %	75,17 %
45-64	2 987	2 558	2 360	198	627	8,39 %	26,57 %
65-74	3 383	2 939	2 754	185	629	6,72 %	22,84 %
75-84	4 268	3 887	3 680	207	588	5,63 %	15,98 %
85+	5 096	4 894	4 727	167	369	3,53 %	7,81 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Maine	**31 282**	**27 616**	**26 845**	**3 666**	**4 437**	**13,27 %**	**16,53 %**
0-24			11		-11		-100,00 %
25-44	1 270	771	629	499	641	64,72 %	101,91 %
45-64	5 545	4 717	4 715	828	830	17,55 %	17,60 %
65-74	6 322	5 417	5 219	905	1 103	16,71 %	21,13 %
75-84	8 155	7 275	6 763	880	1 392	12,10 %	20,58 %
85+	9 990	9 436	9 508	554	482	5,87 %	5,07 %



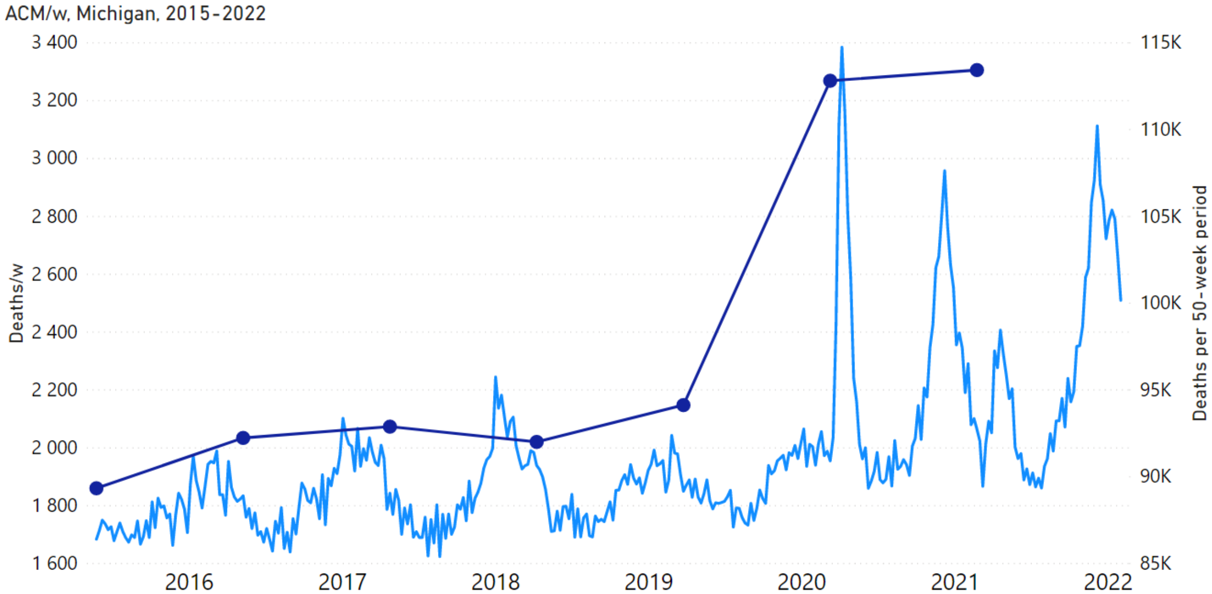
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Maryland	**55 398**	**59 489**	**48 222**	**11 267**	**7 176**	**23,36 %**	**14,88 %**
0-24	1 061	1 095	1 026	69	35	6,73 %	3,41 %
25-44	3 538	3 553	2 902	651	636	22,43 %	21,92 %
45-64	11 118	11 338	9 188	2 150	1 930	23,40 %	21,01 %
65-74	11 105	11 580	9 121	2 459	1 984	26,96 %	21,75 %
75-84	13 114	13 901	11 241	2 660	1 873	23,66 %	16,66 %
85+	15 462	18 022	14 744	3 278	718	22,23 %	4,87 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Maryland	**114 887**	**96 100**	**95 443**	**18 787**	**19 444**	**19,55 %**	**20,37 %**
0-24	2 156	1 996	2 228	160	-72	8,02 %	-3,23 %
25-44	7 091	5 667	5 586	1 424	1 505	25,13 %	26,94 %
45-64	22 456	18 738	19 149	3 718	3 307	19,84 %	17,27 %
65-74	22 685	17 940	17 401	4 745	5 284	26,45 %	30,37 %
75-84	27 015	22 179	21 371	4 836	5 644	21,80 %	26,41 %
85+	33 484	29 580	29 708	3 904	3 776	13,20 %	12,71 %



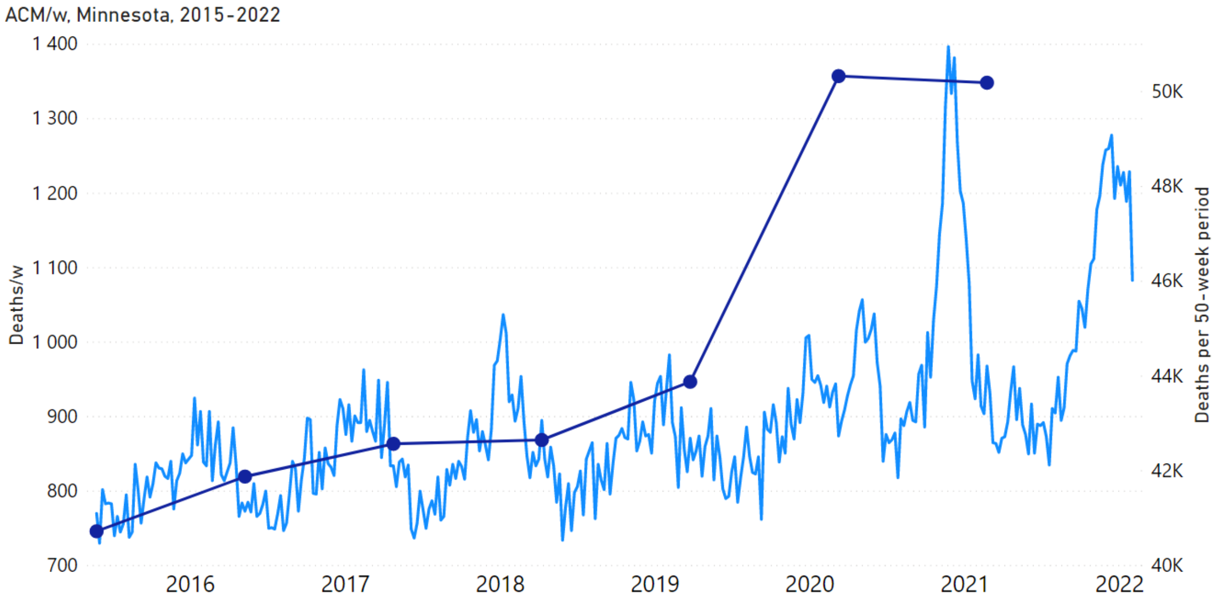
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Massachusetts**	**61 239**	**67 951**	**56 925**	**11 026**	**4 314**	**19,37 %**	**7,58 %**
0-24	727	687	729	-42	-2	-5,76 %	-0,27 %
25-44	3 001	2 947	2 606	341	395	13,09 %	15,16 %
45-64	10 340	10 417	9 103	1 314	1 237	14,43 %	13,59 %
65-74	11 565	12 074	9 874	2 200	1 691	22,28 %	17,13 %
75-84	14 907	16 338	13 233	3 105	1 674	23,46 %	12,65 %
85+	20 699	25 488	21 380	4 108	-681	19,21 %	-3,19 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Massachusetts**	**129 190**	**113 969**	**113 881**	**15 221**	**15 309**	**13,36 %**	**13,44 %**
0-24	1 414	1 517	1 732	-103	-318	-6,79 %	-18,36 %
25-44	5 948	5 178	5 369	770	579	14,87 %	10,78 %
45-64	20 757	18 424	18 431	2 333	2 326	12,66 %	12,62 %
65-74	23 639	19 498	18 994	4 141	4 645	21,24 %	24,46 %
75-84	31 245	26 646	25 754	4 599	5 491	17,26 %	21,32 %
85+	46 187	42 706	43 601	3 481	2 586	8,15 %	5,93 %



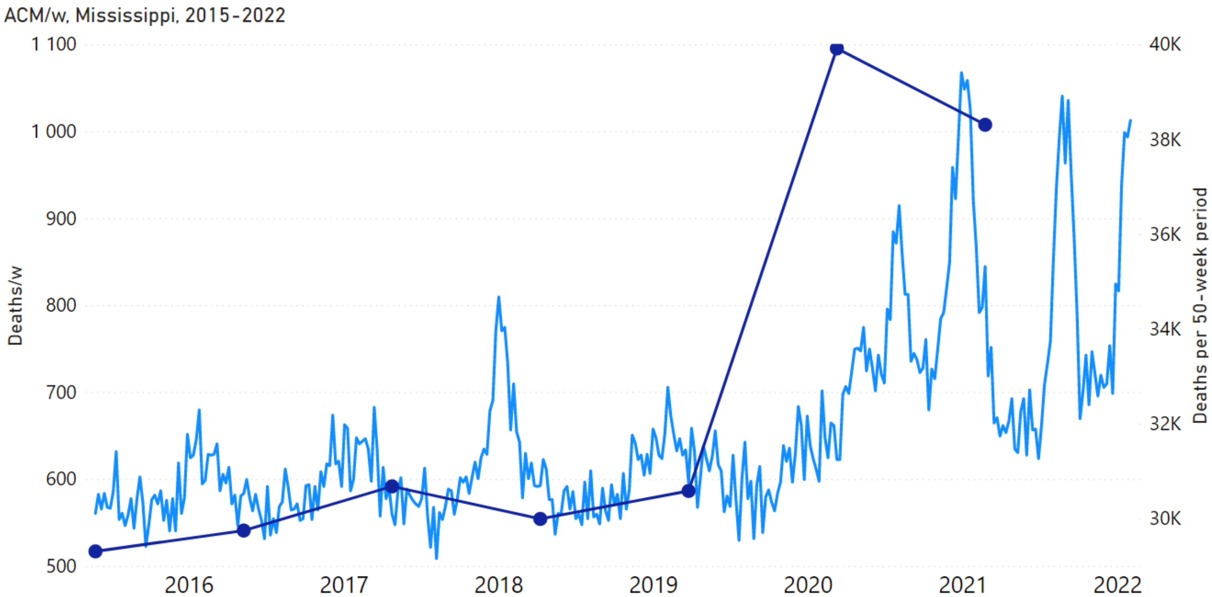
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Michigan**	**113 485**	**112 822**	**94 128**	**18 694**	**19 357**	**19,86 %**	**20,56 %**
0-24	1 934	1 893	1 761	132	173	7,50 %	9,82 %
25-44	6 025	5 275	4 220	1 055	1 805	25,00 %	42,77 %
45-64	22 338	20 541	17 338	3 203	5 000	18,47 %	28,84 %
65-74	24 433	23 162	18 759	4 403	5 674	23,47 %	30,25 %
75-84	27 303	27 635	22 510	5 125	4 793	22,77 %	21,29 %
85+	31 452	34 316	29 540	4 776	1 912	16,17 %	6,47 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Michigan**	**226 307**	**186 135**	**185 124**	**40 172**	**41 183**	**21,58 %**	**22,25 %**
0-24	3 827	3 545	3 925	282	-98	7,95 %	-2,50 %
25-44	11 300	8 506	8 678	2 794	2 622	32,85 %	30,21 %
45-64	42 879	34 529	35 867	8 350	7 012	24,18 %	19,55 %
65-74	47 595	36 566	34 791	11 029	12 804	30,16 %	36,80 %
75-84	54 938	44 486	42 235	10 452	12 703	23,50 %	30,08 %
85+	65 768	58 503	59 628	7 265	6 140	12,42 %	10,30 %



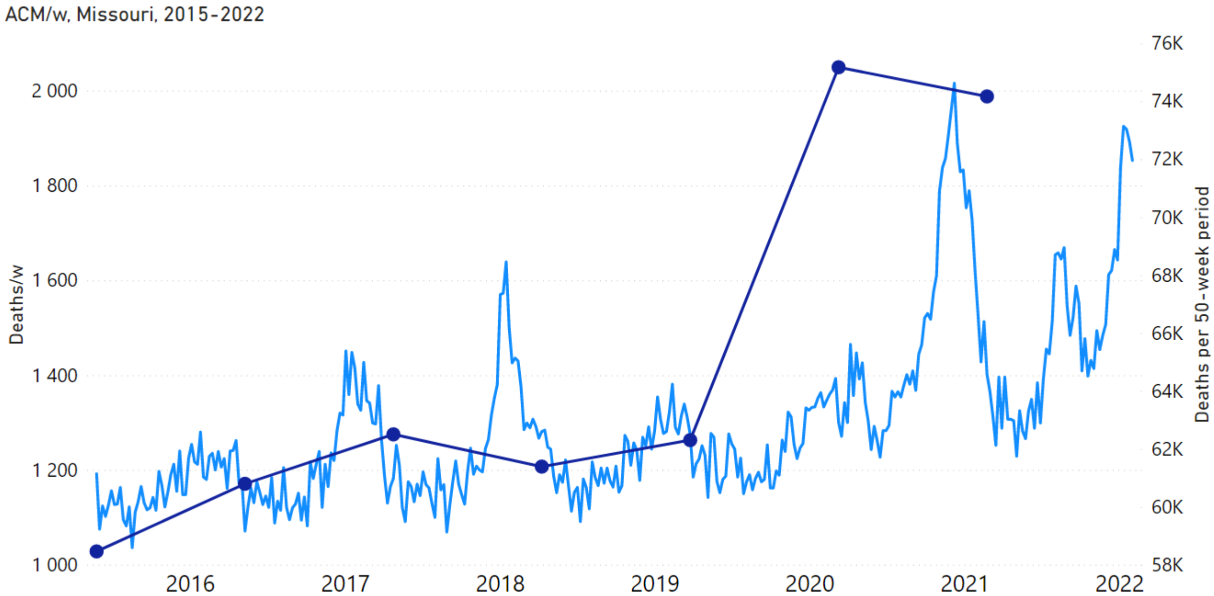
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Minnesota**	**50 304**	**50 330**	**43 879**	**6 451**	**6 425**	**14,70 %**	**14,64 %**
0-24	956	863	873	-10	83	-1,15 %	9,51 %
25-44	2 605	2 192	1 797	395	808	21,98 %	44,96 %
45-64	8 590	7 715	6 792	923	1 798	13,59 %	26,47 %
65-74	9 716	8 881	7 725	1 156	1 991	14,96 %	25,77 %
75-84	12 172	12 487	10 566	1 921	1 606	18,18 %	15,20 %
85+	16 265	18 192	16 126	2 066	139	12,81 %	0,86 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Minnesota**	**100 634**	**86 529**	**84 447**	**14 105**	**16 187**	**16,30 %**	**19,17 %**
0-24	1 819	1 680	1 644	139	175	8,27 %	10,64 %
25-44	4 797	3 401	3 117	1 396	1 680	41,05 %	53,90 %
45-64	16 305	13 655	13 814	2 650	2 491	19,41 %	18,03 %
65-74	18 597	15 115	14 132	3 482	4 465	23,04 %	31,59 %
75-84	24 659	20 714	19 494	3 945	5 165	19,05 %	26,50 %
85+	34 457	31 964	32 246	2 493	2 211	7,80 %	6,86 %



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Mississippi	**38 329**	**39 923**	**30 592**	**9 331**	**7 737**	**30,50 %**	**25,29 %**
0-24	1 013	851	746	105	267	14,08 %	35,79 %
25-44	2 674	2 399	1 776	623	898	35,08 %	50,56 %
45-64	9 130	8 814	6 798	2 016	2 332	29,66 %	34,30 %
65-74	8 851	9 048	6 668	2 380	2 183	35,69 %	32,74 %
75-84	8 943	9 738	7 464	2 274	1 479	30,47 %	19,82 %
85+	7 718	9 073	7 140	1 933	578	27,07 %	8,10 %

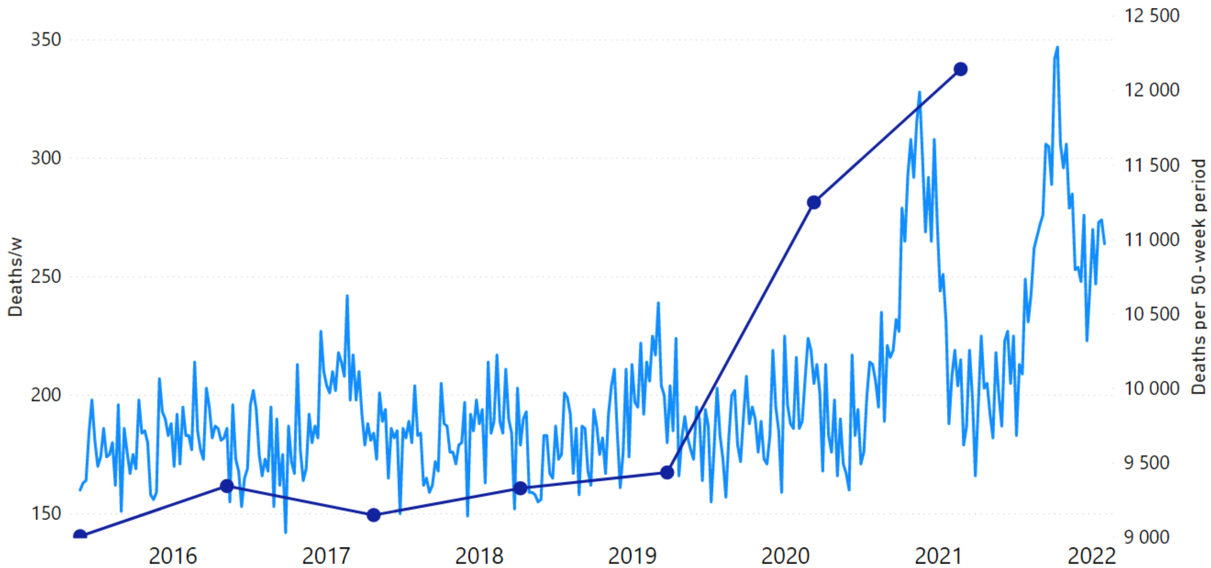
State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Mississippi	**78 252**	**60 594**	**60 446**	**17 658**	**17 806**	**29,14 %**	**29,46 %**
0-24	1 864	1 490	1 434	374	430	25,10 %	29,99 %
25-44	5 073	3 427	3 318	1 646	1 755	48,03 %	52,89 %
45-64	17 944	13 636	14 150	4 308	3 794	31,59 %	26,81 %
65-74	17 899	13 154	12 615	4 745	5 284	36,07 %	41,89 %
75-84	18 681	14 636	14 282	4 045	4 399	27,64 %	30,80 %
85+	16 791	14 251	14 647	2 540	2 144	17,82 %	14,64 %



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Missouri**	**74 286**	**75 191**	**62 319**	**12 872**	**11 967**	**20,66 %**	**19,20 %**
0-24	1 608	1 631	1 524	107	84	7,02 %	5,51 %
25-44	4 529	4 051	3 355	696	1 174	20,75 %	34,99 %
45-64	15 652	13 917	12 090	1 827	3 562	15,11 %	29,46 %
65-74	16 015	15 360	12 592	2 768	3 423	21,98 %	27,18 %
75-84	17 690	18 724	15 041	3 683	2 649	24,49 %	17,61 %
85+	18 792	21 508	17 717	3 791	1 075	21,40 %	6,07 %

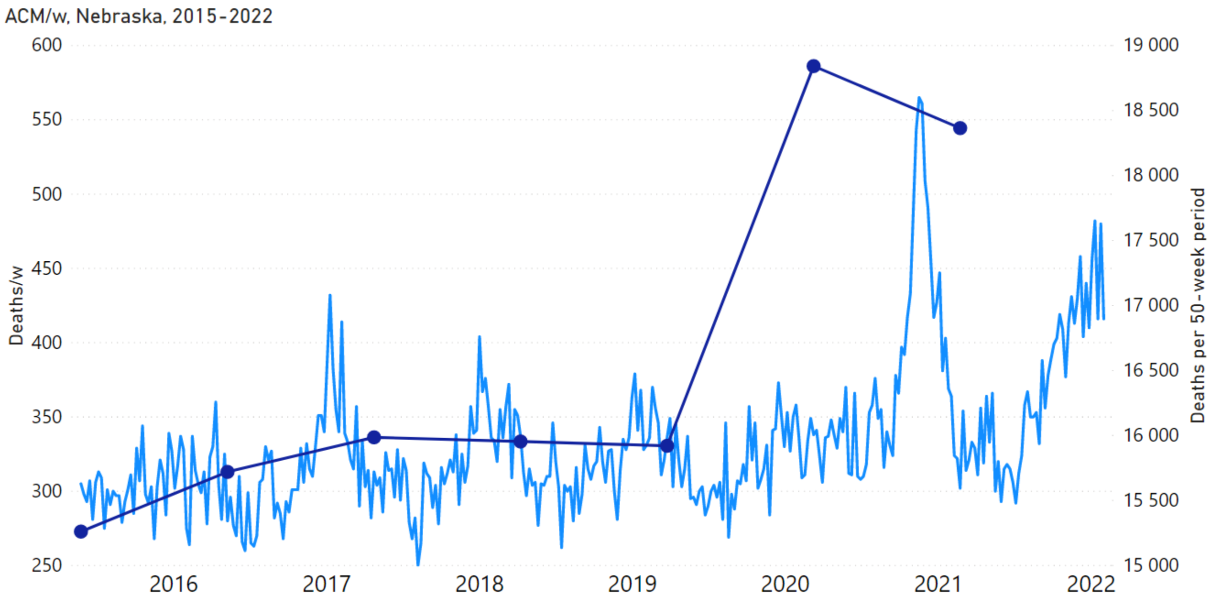
State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Missouri**	**149 477**	**123 724**	**123 330**	**25 753**	**26 147**	**20,81 %**	**21,20 %**
0-24	3 239	3 070	3 194	169	45	5,50 %	1,41 %
25-44	8 580	6 638	6 249	1 942	2 331	29,26 %	37,30 %
45-64	29 569	24 354	24 210	5 215	5 359	21,41 %	22,14 %
65-74	31 375	24 781	23 848	6 594	7 527	26,61 %	31,56 %
75-84	36 414	29 894	29 590	6 520	6 824	21,81 %	23,06 %
85+	40 300	34 987	36 239	5 313	4 061	15,19 %	11,21 %

ACM/w, Montana, 2015-2022



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Montana**	**12 146**	**11 250**	**9 436**	**1 814**	**2 710**	**19,22 %**	**28,72 %**
0-24		11		11		Infinity	
25-44	581	400	193	207	388	107,25 %	201,04 %
45-64	2 306	1 990	1 667	323	639	19,38 %	38,33 %
65-74	2 663	2 366	1 979	387	684	19,56 %	34,56 %
75-84	3 220	2 948	2 520	428	700	16,98 %	27,78 %
85+	3 376	3 535	3 077	458	299	14,88 %	9,72 %

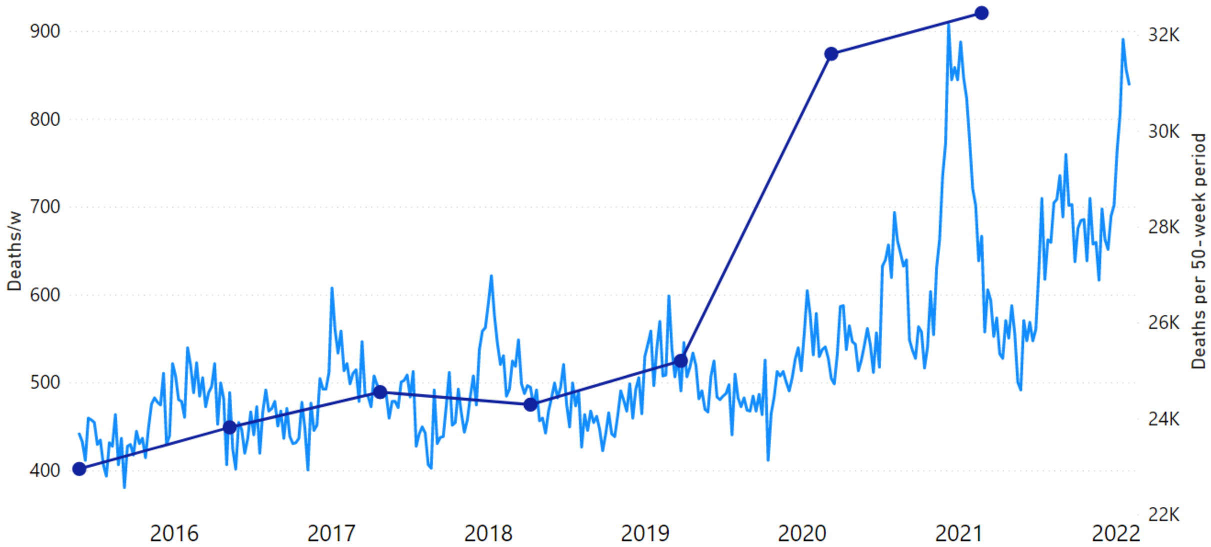
State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Montana**	**23 396**	**18 766**	**18 495**	**4 630**	**4 901**	**24,67 %**	**26,50 %**
0-24	11			11	11	Infinity	Infinity
25-44	981	406	360	575	621	141,63 %	172,50 %
45-64	4 296	3 366	3 414	930	882	27,63 %	25,83 %
65-74	5 029	3 924	3 672	1 105	1 357	28,16 %	36,96 %
75-84	6 168	4 942	4 767	1 226	1 401	24,81 %	29,39 %
85+	6 911	6 128	6 282	783	629	12,78 %	10,01 %



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Nebraska	**18 374**	**18 842**	**15 921**	**2 921**	**2 453**	**18,35 %**	**15,41 %**
0-24	172	126	61	65	111	106,56 %	181,97 %
25-44	761	710	458	252	303	55,02 %	66,16 %
45-64	3 315	3 067	2 698	369	617	13,68 %	22,87 %
65-74	3 792	3 671	3 003	668	789	22,24 %	26,27 %
75-84	4 430	4 713	3 948	765	482	19,38 %	12,21 %
85+	5 904	6 555	5 753	802	151	13,94 %	2,62 %

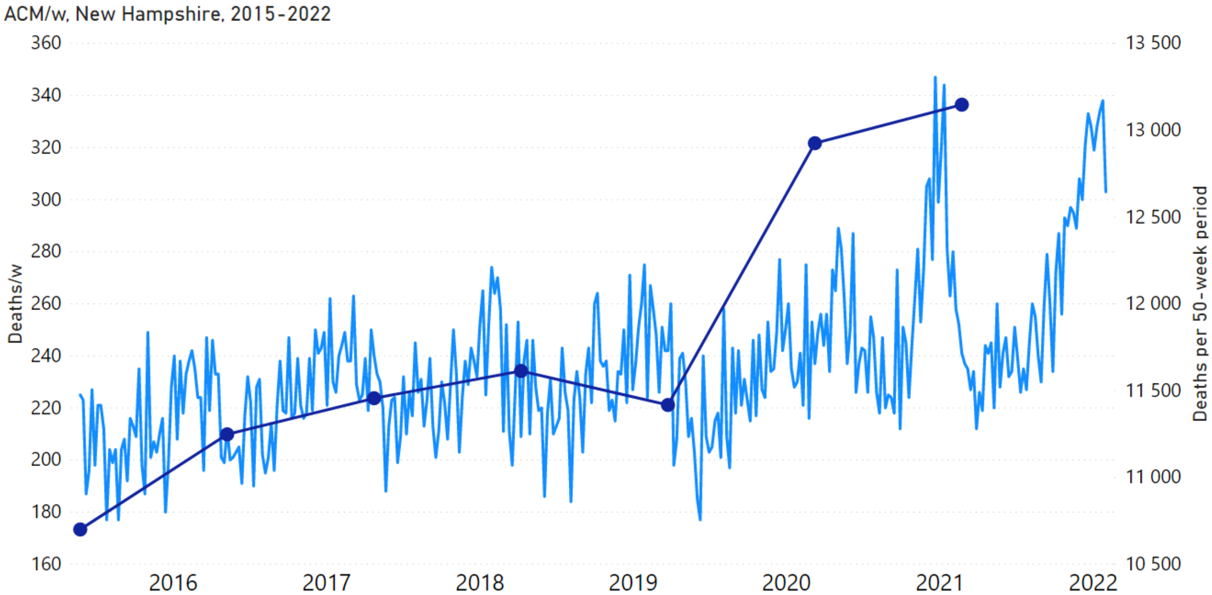
State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Nebraska	**37 216**	**31 875**	**31 706**	**5 341**	**5 510**	**16,76 %**	**17,38 %**
0-24	298	160	214	138	84	86,25 %	39,25 %
25-44	1 471	1 011	913	460	558	45,50 %	61,12 %
45-64	6 382	5 438	5 388	944	994	17,36 %	18,45 %
65-74	7 463	5 958	5 647	1 505	1 816	25,26 %	32,16 %
75-84	9 143	7 792	7 651	1 351	1 492	17,34 %	19,50 %
85+	12 459	11 516	11 893	943	566	8,19 %	4,76 %

ACM/w, Nevada, 2015-2022



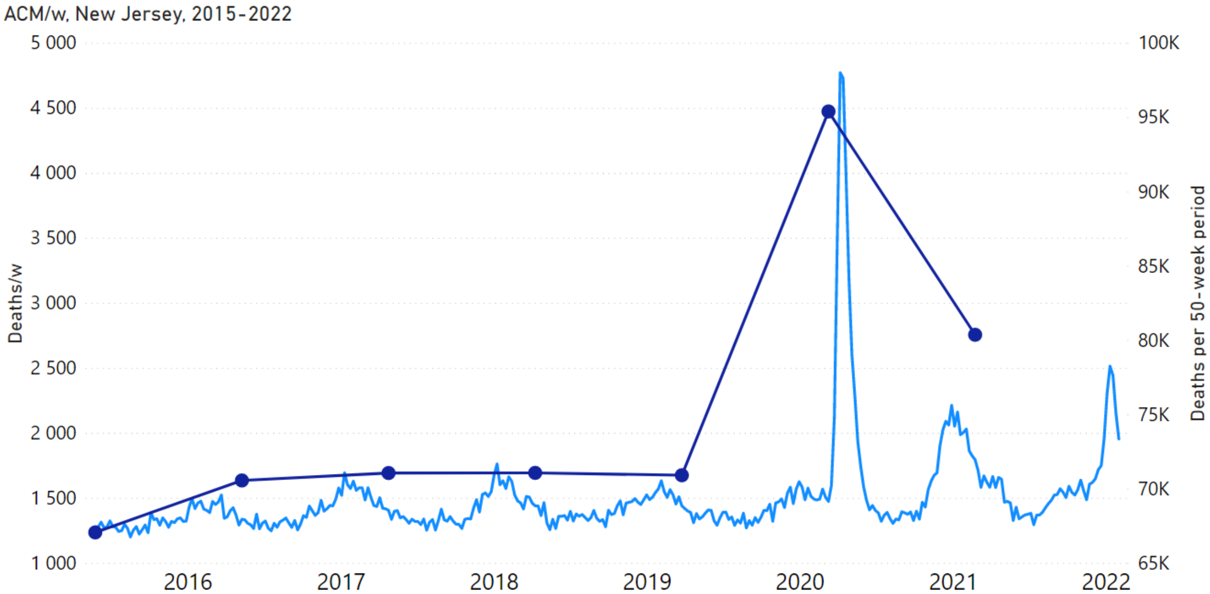
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Nevada	**32 484**	**31 616**	**25 212**	**6 404**	**7 272**	**25,40 %**	**28,84 %**
0-24	558	484	378	106	180	28,04 %	47,62 %
25-44	2 190	1 843	1 337	506	853	37,85 %	63,80 %
45-64	7 202	6 642	5 316	1 326	1 886	24,94 %	35,48 %
65-74	7 720	7 342	5 791	1 551	1 929	26,78 %	33,31 %
75-84	8 375	8 343	6 564	1 779	1 811	27,10 %	27,59 %
85+	6 439	6 962	5 826	1 136	613	19,50 %	10,52 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Nevada	**64 100**	**49 510**	**48 384**	**14 590**	**15 716**	**29,47 %**	**32,48 %**
0-24	1 042	667	817	375	225	56,22 %	27,54 %
25-44	4 033	2 734	2 749	1 299	1 284	47,51 %	46,71 %
45-64	13 844	10 562	10 650	3 282	3 194	31,07 %	29,99 %
65-74	15 062	11 470	11 050	3 592	4 012	31,32 %	36,31 %
75-84	16 718	12 803	12 084	3 915	4 634	30,58 %	38,35 %
85+	13 401	11 274	11 034	2 127	2 367	18,87 %	21,45 %



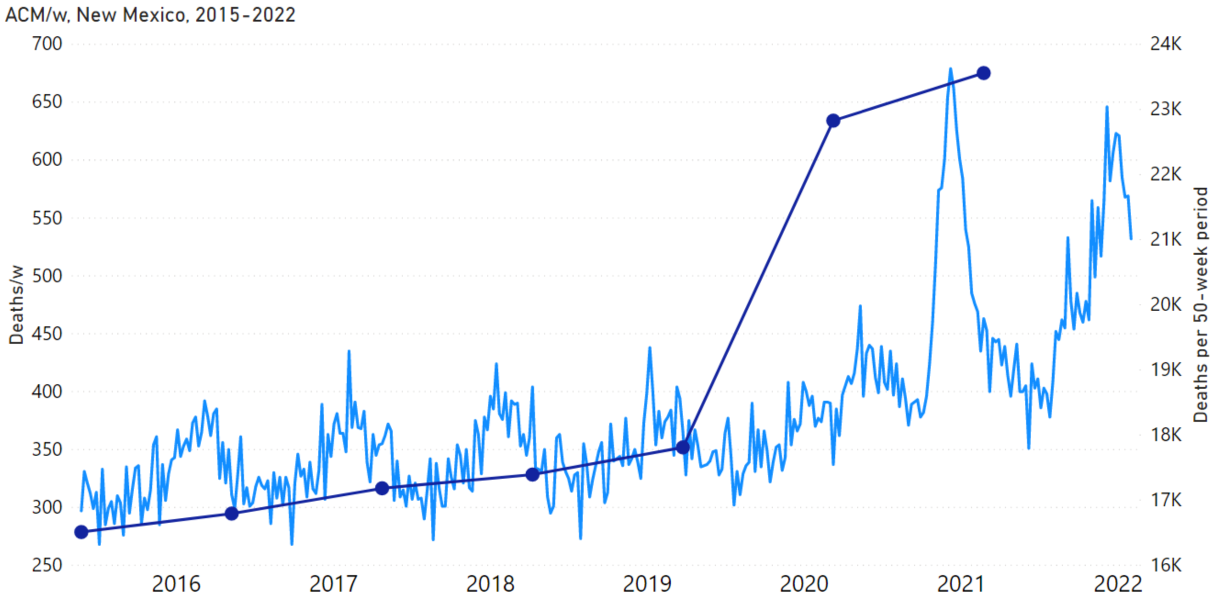
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **New Hampshire**	**13 154**	**12 925**	**11 417**	**1 508**	**1 737**	**13,21 %**	**15,21 %**
25-44	586	345	267	78	319	29,21 %	119,48 %
45-64	2 333	2 048	2 043	5	290	0,24 %	14,19 %
65-74	2 728	2 530	2 190	340	538	15,53 %	24,57 %
75-84	3 393	3 299	2 887	412	506	14,27 %	17,53 %
85+	4 114	4 703	4 030	673	84	16,70 %	2,08 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **New Hampshire**	**26 079**	**23 030**	**22 704**	**3 049**	**3 375**	**13,24 %**	**14,87 %**
25-44	931	584	755	347	176	59,42 %	23,31 %
45-64	4 381	4 126	4 137	255	244	6,18 %	5,90 %
65-74	5 258	4 385	4 108	873	1 150	19,91 %	27,99 %
75-84	6 692	5 739	5 548	953	1 144	16,61 %	20,62 %
85+	8 817	8 196	8 156	621	661	7,58 %	8,10 %



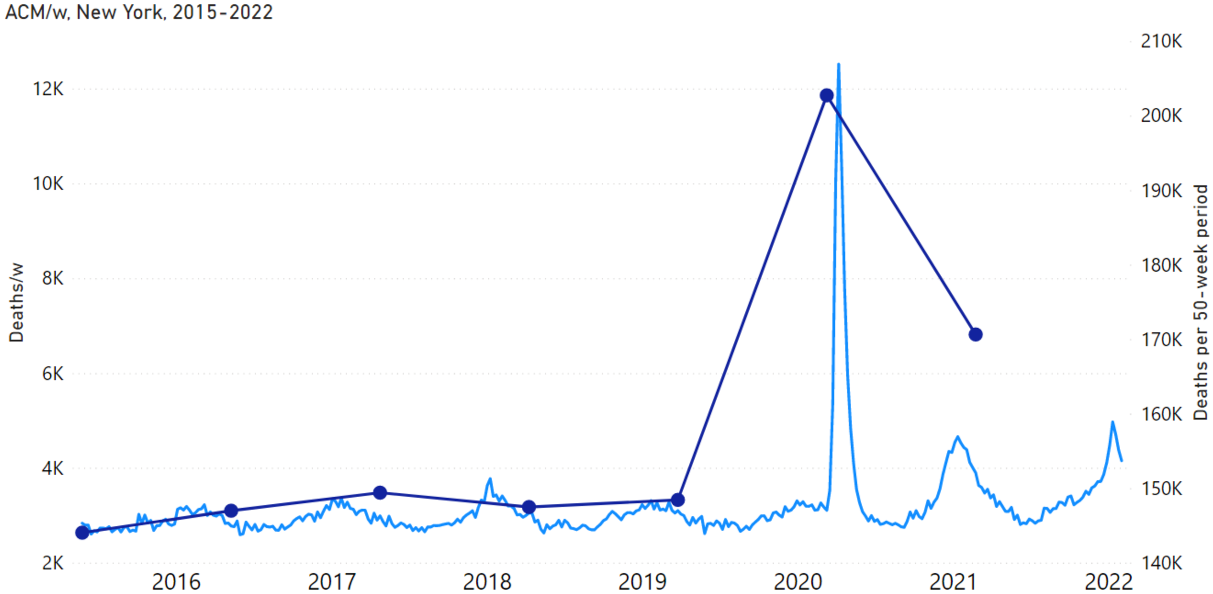
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
New Jersey	**80 676**	**95 415**	**70 933**	**24 482**	**9 743**	**34,51 %**	**13,74 %**
0-24	959	973	957	16	2	1,67 %	0,21 %
25-44	3 959	4 161	3 272	889	687	27,17 %	21,00 %
45-64	14 526	16 878	11 741	5 137	2 785	43,75 %	23,72 %
65-74	15 154	17 563	12 243	5 320	2 911	43,45 %	23,78 %
75-84	19 414	23 160	16 787	6 373	2 627	37,96 %	15,65 %
85+	26 664	32 680	25 933	6 747	731	26,02 %	2,82 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
New Jersey	**176 091**	**142 025**	**141 664**	**34 066**	**34 427**	**23,99 %**	**24,30 %**
0-24	1 932	1 984	2 186	-52	-254	-2,62 %	-11,62 %
25-44	8 120	6 602	6 392	1 518	1 728	22,99 %	27,03 %
45-64	31 404	23 852	24 287	7 552	7 117	31,66 %	29,30 %
65-74	32 717	24 423	23 526	8 294	9 191	33,96 %	39,07 %
75-84	42 574	33 345	32 755	9 229	9 819	27,68 %	29,98 %
85+	59 344	51 819	52 518	7 525	6 826	14,52 %	13,00 %



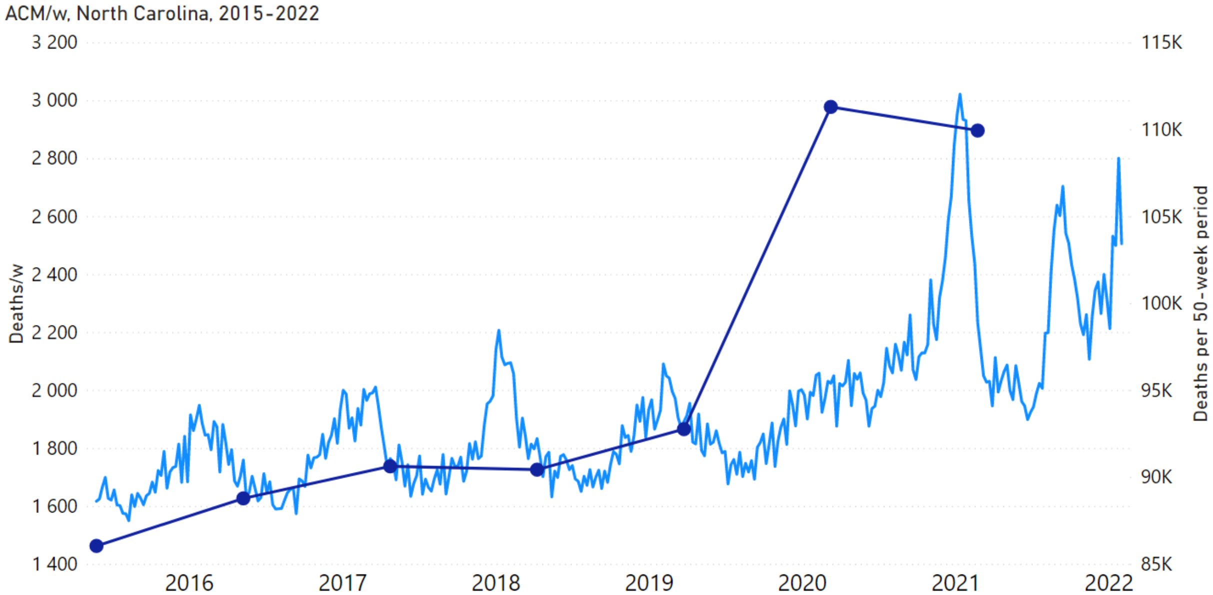
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
New Mexico	**23 619**	**22 829**	**17 810**	**5 019**	**5 809**	**28,18 %**	**32,62 %**
0-24	357	200	206	-6	151	-2,91 %	73,30 %
25-44	2 459	2 025	1 478	547	981	37,01 %	66,37 %
45-64	5 237	4 771	3 656	1 115	1 581	30,50 %	43,24 %
65-74	4 826	4 644	3 489	1 155	1 337	33,10 %	38,32 %
75-84	5 176	5 397	4 142	1 255	1 034	30,30 %	24,96 %
85+	5 564	5 792	4 839	953	725	19,69 %	14,98 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
New Mexico	**46 448**	**35 203**	**33 976**	**11 245**	**12 472**	**31,94 %**	**36,71 %**
0-24	557	352	407	205	150	58,24 %	36,86 %
25-44	4 484	2 772	2 476	1 712	2 008	61,76 %	81,10 %
45-64	10 008	7 181	7 105	2 827	2 903	39,37 %	40,86 %
65-74	9 470	7 021	6 628	2 449	2 842	34,88 %	42,88 %
75-84	10 573	8 184	7 836	2 389	2 737	29,19 %	34,93 %
85+	11 356	9 693	9 524	1 663	1 832	17,16 %	19,24 %



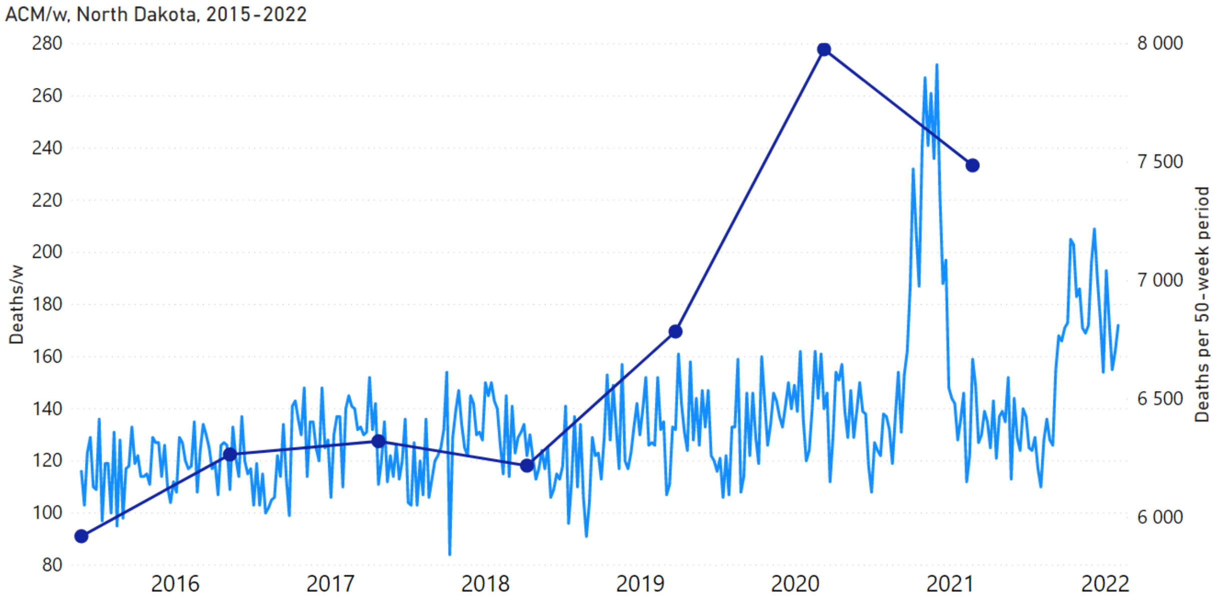
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **New York**	**170 873**	**202 806**	**148 501**	**54 305**	**22 372**	**36,57 %**	**15,07 %**
0-24	2 482	2 498	2 441	57	41	2,34 %	1,68 %
25-44	8 644	8 910	6 342	2 568	2 302	40,49 %	36,30 %
45-64	31 464	35 813	25 388	10 425	6 076	41,06 %	23,93 %
65-74	33 371	38 953	27 197	11 756	6 174	43,23 %	22,70 %
75-84	40 363	48 635	34 947	13 688	5 416	39,17 %	15,50 %
85+	54 549	67 997	52 186	15 811	2 363	30,30 %	4,53 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **New York**	**373 679**	**296 046**	**296 515**	**77 633**	**77 164**	**26,22 %**	**26,02 %**
0-24	4 980	4 877	5 261	103	-281	2,11 %	-5,34 %
25-44	17 554	12 381	12 444	5 173	5 110	41,78 %	41,06 %
45-64	67 277	51 057	52 583	16 220	14 694	31,77 %	27,94 %
65-74	72 324	53 676	52 079	18 648	20 245	34,74 %	38,87 %
75-84	88 998	69 598	67 835	19 400	21 163	27,87 %	31,20 %
85+	122 546	104 457	106 313	18 089	16 233	17,32 %	15,27 %



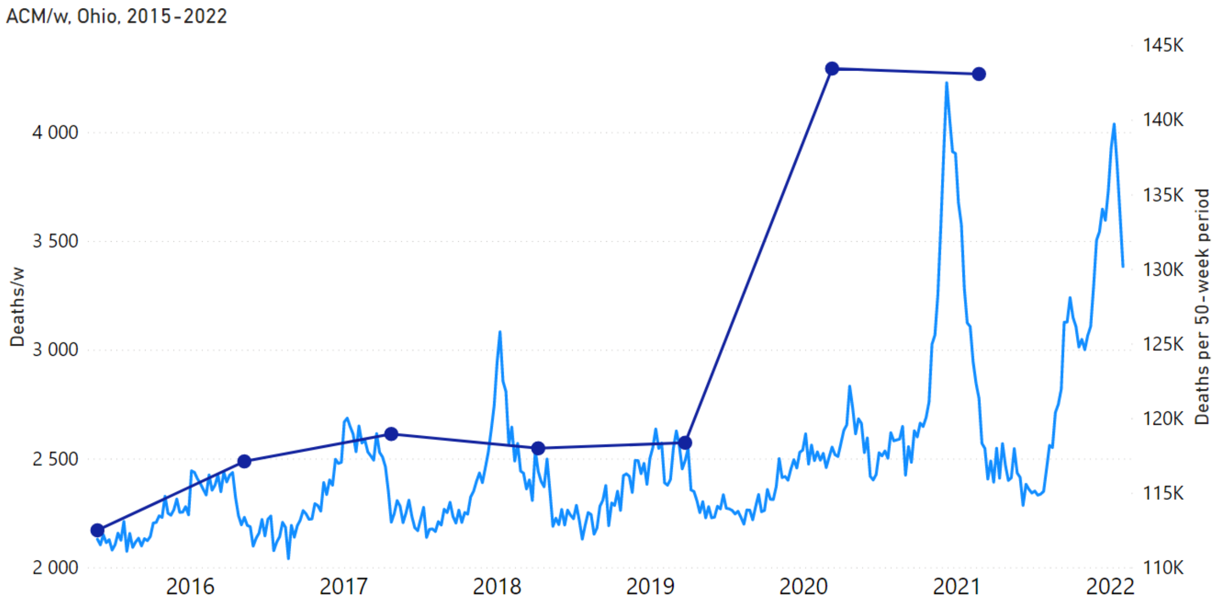
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
North Carolina	**111 328**	**111 321**	**92 780**	**18 541**	**18 548**	**19,98 %**	**19,99 %**
0-24	2 450	2 432	2 129	303	321	14,23 %	15,08 %
25-44	7 240	6 267	4 851	1 416	2 389	29,19 %	49,25 %
45-64	23 868	21 405	18 146	3 259	5 722	17,96 %	31,53 %
65-74	24 093	23 316	19 145	4 171	4 948	21,79 %	25,84 %
75-84	26 905	28 108	23 053	5 055	3 852	21,93 %	16,71 %
85+	26 772	29 793	25 456	4 337	1 316	17,04 %	5,17 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
North Carolina	**222 649**	**183 230**	**179 440**	**39 419**	**43 209**	**21,51 %**	**24,08 %**
0-24	4 882	4 213	4 383	669	499	15,88 %	11,38 %
25-44	13 507	9 552	9 201	3 955	4 306	41,40 %	46,80 %
45-64	45 273	36 207	36 731	9 066	8 542	25,04 %	23,26 %
65-74	47 409	37 821	36 709	9 588	10 700	25,35 %	29,15 %
75-84	55 013	45 397	42 877	9 616	12 136	21,18 %	28,30 %
85+	56 565	50 040	49 539	6 525	7 026	13,04 %	14,18 %



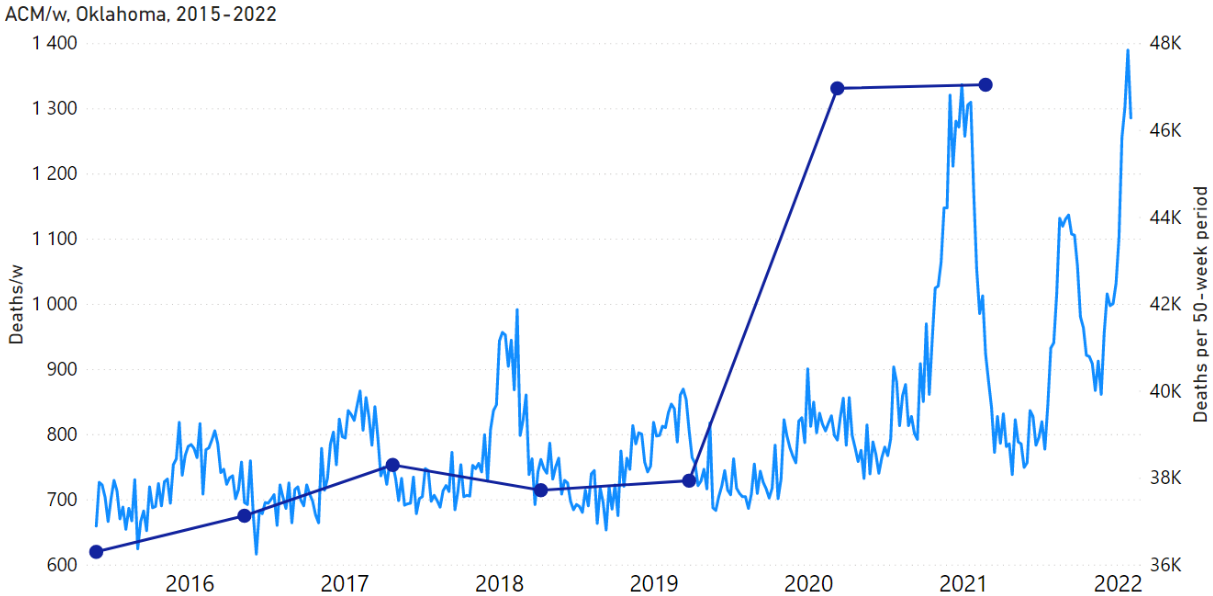
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **North Dakota**	**7 601**	**7 977**	**6 786**	**1 191**	**815**	**17,55 %**	**12,01 %**
25-44	328	183	112	71	216	63,39 %	192,86 %
45-64	1 535	1 332	1 231	101	304	8,20 %	24,70 %
65-74	1 612	1 496	1 260	236	352	18,73 %	27,94 %
75-84	1 817	1 987	1 656	331	161	19,99 %	9,72 %
85+	2 309	2 979	2 527	452	-218	17,89 %	-8,63 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **North Dakota**	**15 578**	**13 006**	**12 590**	**2 572**	**2 988**	**19,78 %**	**23,73 %**
0-24			13		-13		-100,00 %
25-44	511	159	106	352	405	221,38 %	382,08 %
45-64	2 867	2 388	2 232	479	635	20,06 %	28,45 %
65-74	3 108	2 420	2 206	688	902	28,43 %	40,89 %
75-84	3 804	3 169	3 101	635	703	20,04 %	22,67 %
85+	5 288	4 870	4 932	418	356	8,58 %	7,22 %



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Ohio**	**143 192**	**143 472**	**118 385**	**25 087**	**24 807**	**21,19 %**	**20,95 %**
0-24	2 657	2 511	2 379	132	278	5,55 %	11,69 %
25-44	8 147	7 364	6 125	1 239	2 022	20,23 %	33,01 %
45-64	28 907	25 907	22 227	3 680	6 680	16,56 %	30,05 %
65-74	31 158	28 861	23 323	5 538	7 835	23,74 %	33,59 %
75-84	34 083	35 299	28 731	6 568	5 352	22,86 %	18,63 %
85+	38 240	43 530	35 600	7 930	2 640	22,28 %	7,42 %

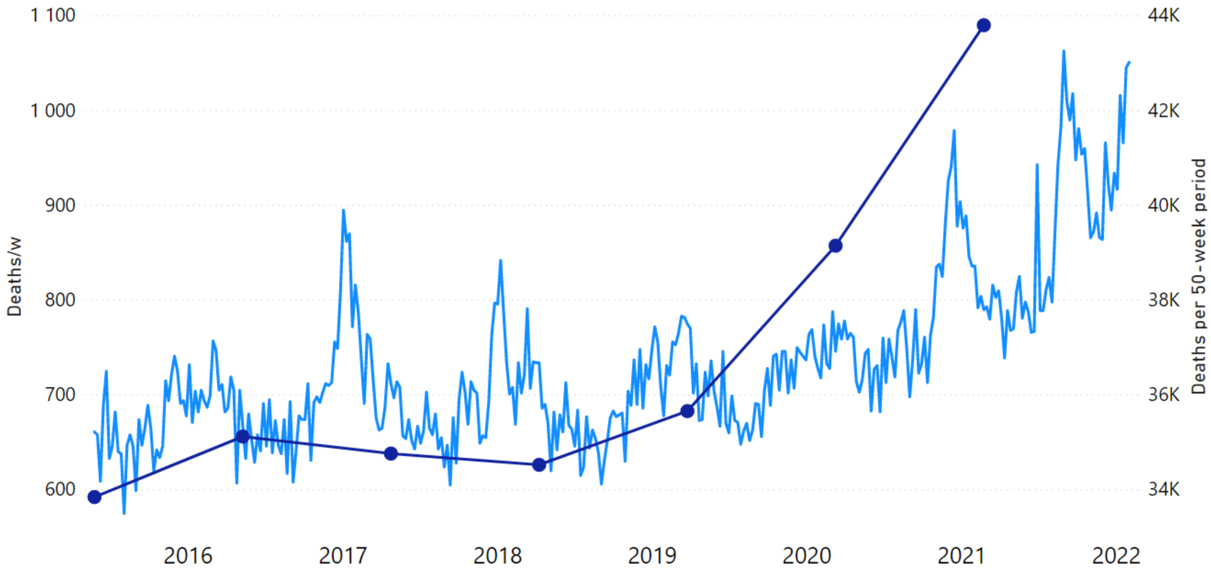
State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Ohio**	**286 664**	**236 399**	**236 108**	**50 265**	**50 556**	**21,26 %**	**21,41 %**
0-24	5 168	4 778	5 452	390	-284	8,16 %	-5,21 %
25-44	15 511	12 105	12 705	3 406	2 806	28,14 %	22,09 %
45-64	54 814	44 740	45 701	10 074	9 113	22,52 %	19,94 %
65-74	60 019	46 070	43 912	13 949	16 107	30,28 %	36,68 %
75-84	69 382	56 900	55 164	12 482	14 218	21,94 %	25,77 %
85+	81 770	71 806	73 174	9 964	8 596	13,88 %	11,75 %



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Oklahoma	**47 131**	**46 971**	**37 943**	**9 028**	**9 188**	**23,79 %**	**24,22 %**
0-24	1 002	905	837	68	165	8,12 %	19,71 %
25-44	2 909	2 409	1 935	474	974	24,50 %	50,34 %
45-64	10 477	9 434	7 957	1 477	2 520	18,56 %	31,67 %
65-74	10 624	10 359	8 124	2 235	2 500	27,51 %	30,77 %
75-84	11 575	11 815	9 473	2 342	2 102	24,72 %	22,19 %
85+	10 544	12 049	9 617	2 432	927	25,29 %	9,64 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Oklahoma	**94 102**	**75 667**	**75 441**	**18 435**	**18 661**	**24,36 %**	**24,74 %**
0-24	1 907	1 702	1 901	205	6	12,04 %	0,32 %
25-44	5 318	3 878	4 011	1 440	1 307	37,13 %	32,59 %
45-64	19 911	16 046	16 446	3 865	3 465	24,09 %	21,07 %
65-74	20 983	16 069	15 273	4 914	5 710	30,58 %	37,39 %
75-84	23 390	18 690	18 270	4 700	5 120	25,15 %	28,02 %
85+	22 593	19 282	19 540	3 311	3 053	17,17 %	15,62 %

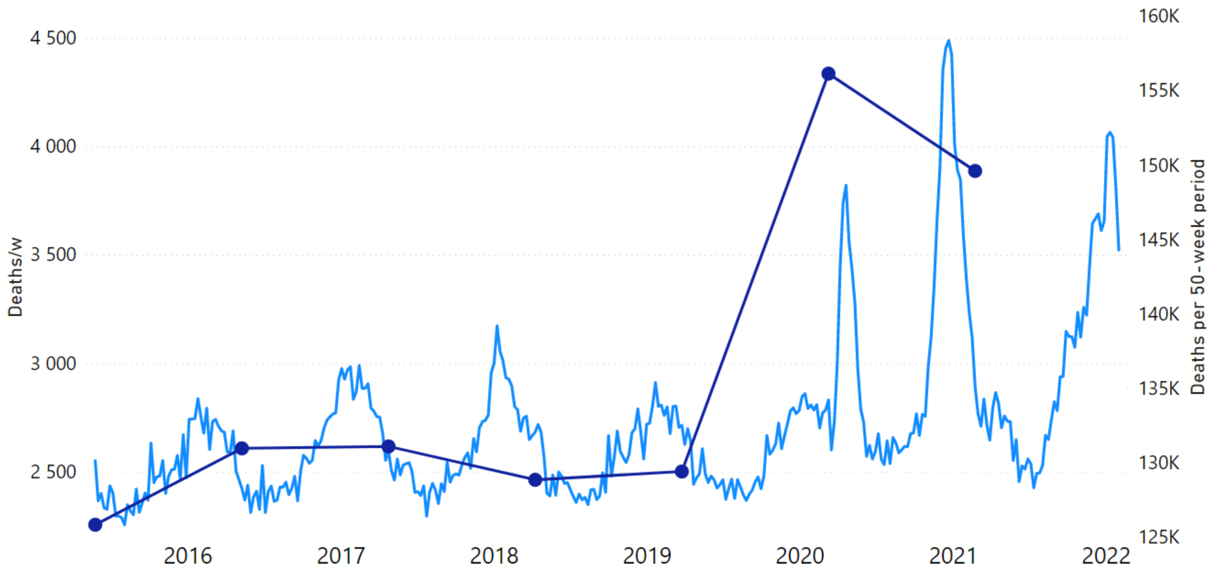
ACM/w, Oregon, 2015-2022



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Oregon**	**43 835**	**39 150**	**35 660**	**3 490**	**8 175**	**9,79 %**	**22,92 %**
0-24	533	563	409	154	124	37,65 %	30,32 %
25-44	2 304	1 775	1 504	271	800	18,02 %	53,19 %
45-64	7 821	6 725	6 044	681	1 777	11,27 %	29,40 %
65-74	9 353	8 141	7 350	791	2 003	10,76 %	27,25 %
75-84	10 998	9 804	8 855	949	2 143	10,72 %	24,20 %
85+	12 826	12 142	11 498	644	1 328	5,60 %	11,55 %

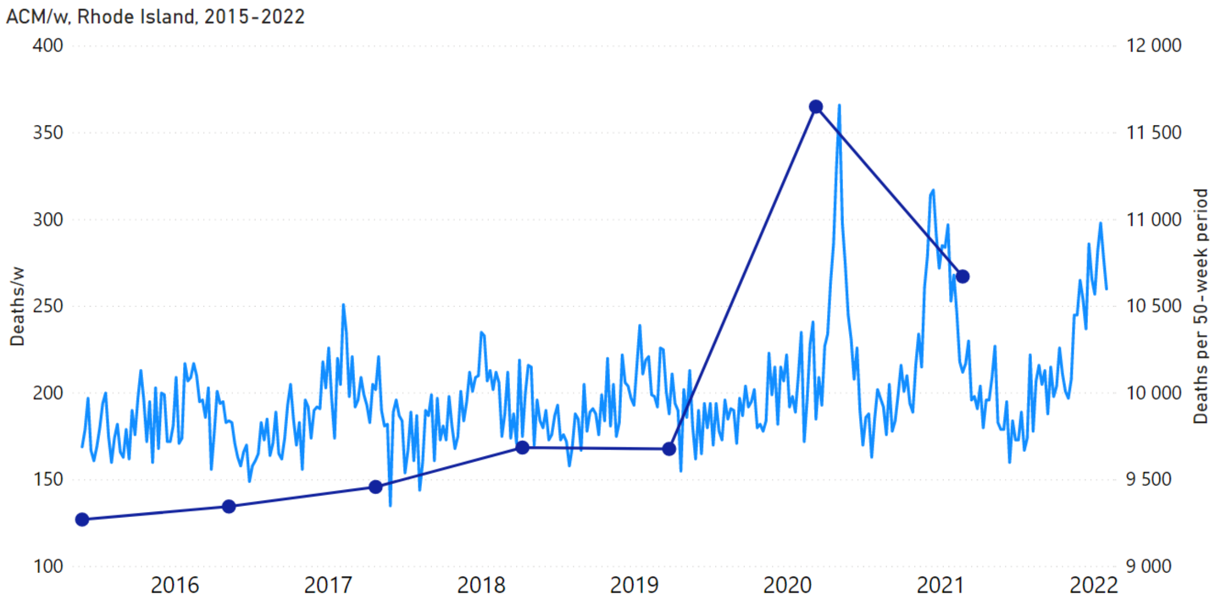
State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Oregon**	**82 985**	**70 186**	**69 886**	**12 799**	**13 099**	**18,24 %**	**18,74 %**
0-24	1 096	810	1 221	286	-125	35,31 %	-10,24 %
25-44	4 079	2 908	2 674	1 171	1 405	40,27 %	52,54 %
45-64	14 546	12 130	12 418	2 416	2 128	19,92 %	17,14 %
65-74	17 494	14 370	13 648	3 124	3 846	21,74 %	28,18 %
75-84	20 802	17 248	16 525	3 554	4 277	20,61 %	25,88 %
85+	24 968	22 720	23 400	2 248	1 568	9,89 %	6,70 %

ACM/w, Pennsylvania, 2015-2022



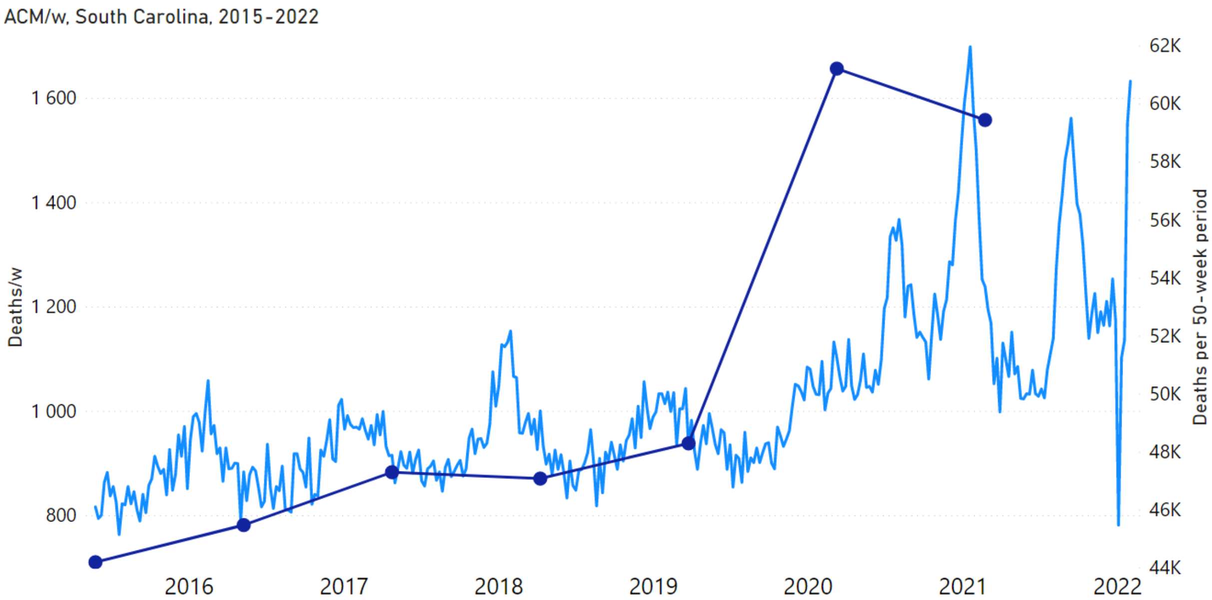
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Pennsylvania	**149 833**	**156 171**	**129 421**	**26 750**	**20 412**	**20,67 %**	**15,77 %**
0-24	2 449	2 306	2 200	106	249	4,82 %	11,32 %
25-44	7 526	6 841	5 952	889	1 574	14,94 %	26,44 %
45-64	27 122	25 193	21 797	3 396	5 325	15,58 %	24,43 %
65-74	30 662	29 558	23 564	5 994	7 098	25,44 %	30,12 %
75-84	36 201	37 980	31 103	6 877	5 098	22,11 %	16,39 %
85+	45 873	54 293	44 805	9 488	1 068	21,18 %	2,38 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Pennsylvania	**306 004**	**258 285**	**262 088**	**47 719**	**43 916**	**18,48 %**	**16,76 %**
0-24	4 755	4 505	5 117	250	-362	5,55 %	-7,07 %
25-44	14 367	11 912	12 488	2 455	1 879	20,61 %	15,05 %
45-64	52 315	43 813	44 916	8 502	7 399	19,41 %	16,47 %
65-74	60 220	46 806	45 686	13 414	14 534	28,66 %	31,81 %
75-84	74 181	61 353	60 729	12 828	13 452	20,91 %	22,15 %
85+	100 166	89 896	93 152	10 270	7 014	11,42 %	7,53 %



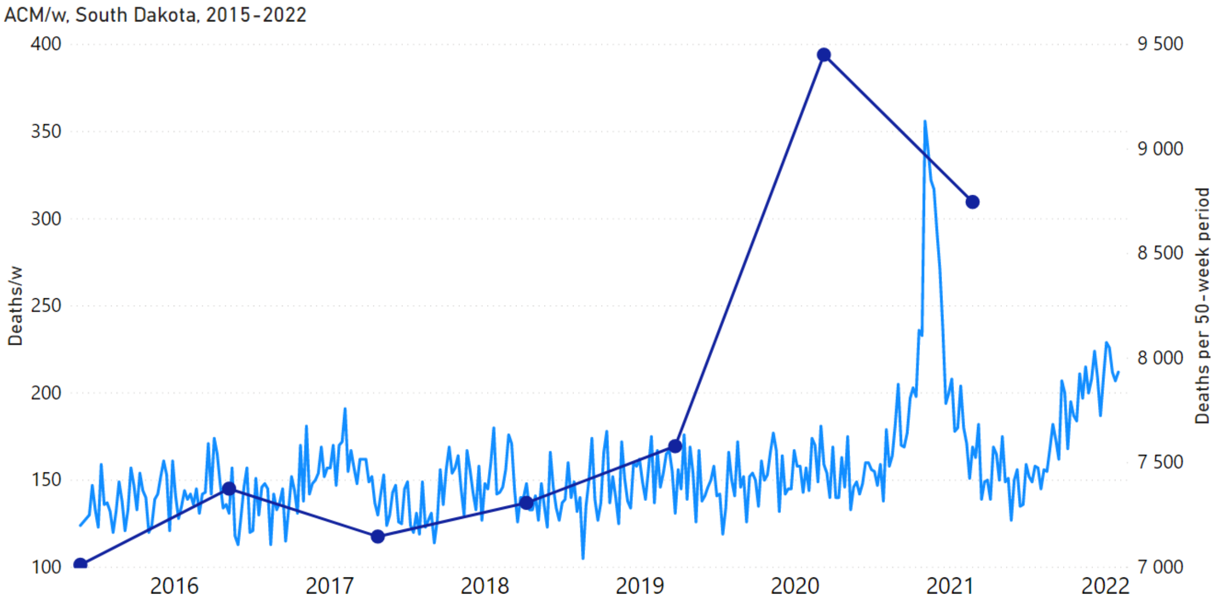
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Rhode Island**	**10 674**	**11 651**	**9 678**	**1 973**	**996**	**20,39 %**	**10,29 %**
25-44	263	195	129	66	134	51,16 %	103,88 %
45-64	1 879	1 875	1 569	306	310	19,50 %	19,76 %
65-74	2 131	2 092	1 677	415	454	24,75 %	27,07 %
75-84	2 607	2 773	2 381	392	226	16,46 %	9,49 %
85+	3 794	4 716	3 922	794	-128	20,24 %	-3,26 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Rhode Island**	**22 325**	**19 364**	**18 805**	**2 961**	**3 520**	**15,29 %**	**18,72 %**
25-44	458	221	223	237	235	107,24 %	105,38 %
45-64	3 754	3 156	3 185	598	569	18,95 %	17,86 %
65-74	4 223	3 496	3 283	727	940	20,80 %	28,63 %
75-84	5 380	4 656	4 269	724	1 111	15,55 %	26,02 %
85+	8 510	7 835	7 845	675	665	8,62 %	8,48 %



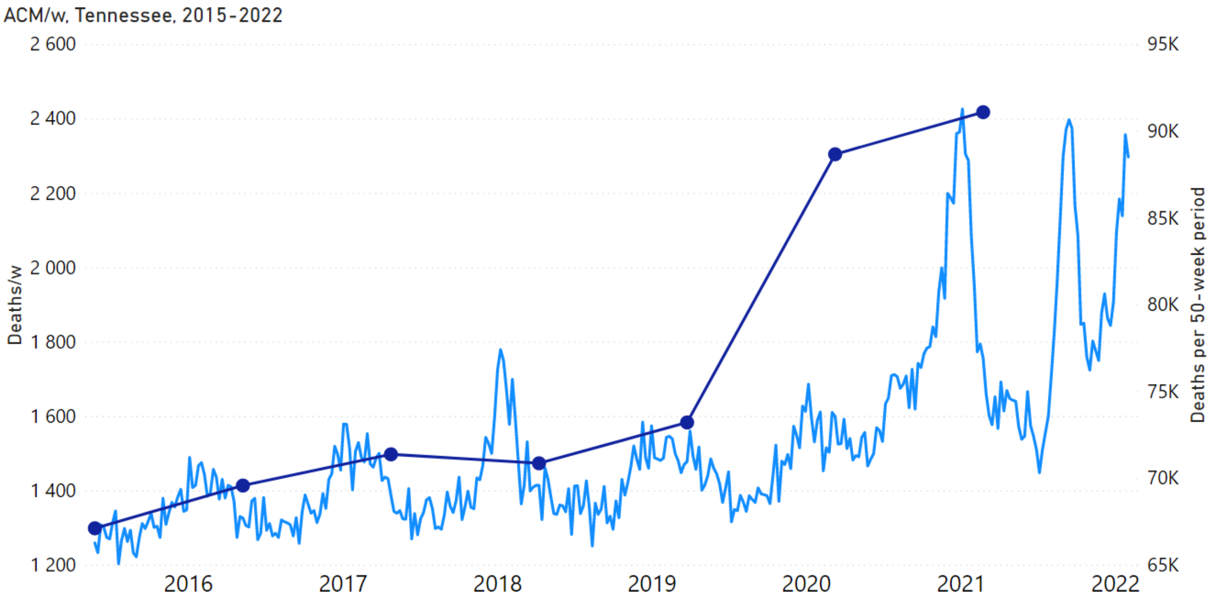
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **South Carolina**	**59 495**	**61 227**	**48 298**	**12 929**	**11 197**	**26,77 %**	**23,18 %**
0-24	1 375	1 251	1 165	86	210	7,38 %	18,03 %
25-44	4 057	3 464	2 689	775	1 368	28,82 %	50,87 %
45-64	13 074	12 394	10 034	2 360	3 040	23,52 %	30,30 %
65-74	13 479	13 421	10 339	3 082	3 140	29,81 %	30,37 %
75-84	14 355	15 568	12 042	3 526	2 313	29,28 %	19,21 %
85+	13 155	15 129	12 029	3 100	1 126	25,77 %	9,36 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **South Carolina**	**120 722**	**95 381**	**92 782**	**25 341**	**27 940**	**26,57 %**	**30,11 %**
0-24	2 626	2 249	2 215	377	411	16,76 %	18,56 %
25-44	7 521	5 306	4 878	2 215	2 643	41,75 %	54,18 %
45-64	25 468	19 904	20 227	5 564	5 241	27,95 %	25,91 %
65-74	26 900	20 460	19 697	6 440	7 203	31,48 %	36,57 %
75-84	29 923	23 582	22 209	6 341	7 714	26,89 %	34,73 %
85+	28 284	23 880	23 556	4 404	4 728	18,44 %	20,07 %



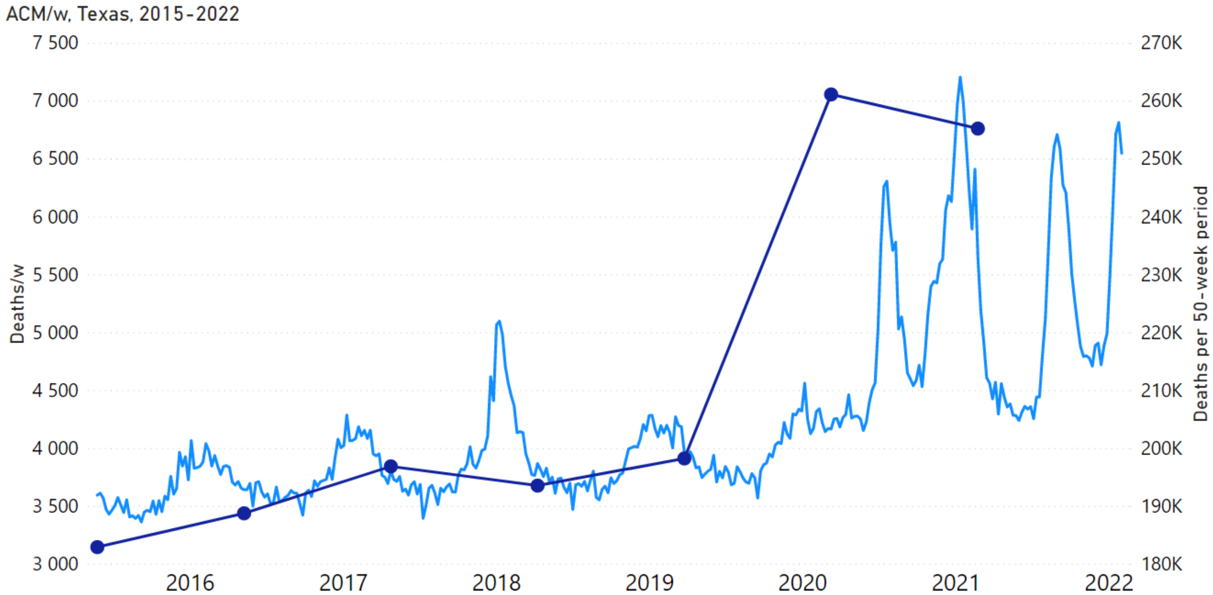
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **South Dakota**	**8 757**	**9 451**	**7 579**	**1 872**	**1 178**	**24,70 %**	**15,54 %**
0-24	11	22		22	11	Infinity	Infinity
25-44	404	226	128	98	276	76,56 %	215,63 %
45-64	1 686	1 624	1 353	271	333	20,03 %	24,61 %
65-74	1 857	1 901	1 410	491	447	34,82 %	31,70 %
75-84	2 035	2 290	1 852	438	183	23,65 %	9,88 %
85+	2 764	3 388	2 836	552	-72	19,46 %	-2,54 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **South Dakota**	**18 208**	**14 887**	**14 523**	**3 321**	**3 685**	**22,31 %**	**25,37 %**
0-24	33		12	33	21	Infinity	175,00 %
25-44	630	173	145	457	485	264,16 %	334,48 %
45-64	3 310	2 709	2 626	601	684	22,19 %	26,05 %
65-74	3 758	2 817	2 630	941	1 128	33,40 %	42,89 %
75-84	4 325	3 605	3 495	720	830	19,97 %	23,75 %
85+	6 152	5 583	5 615	569	537	10,19 %	9,56 %



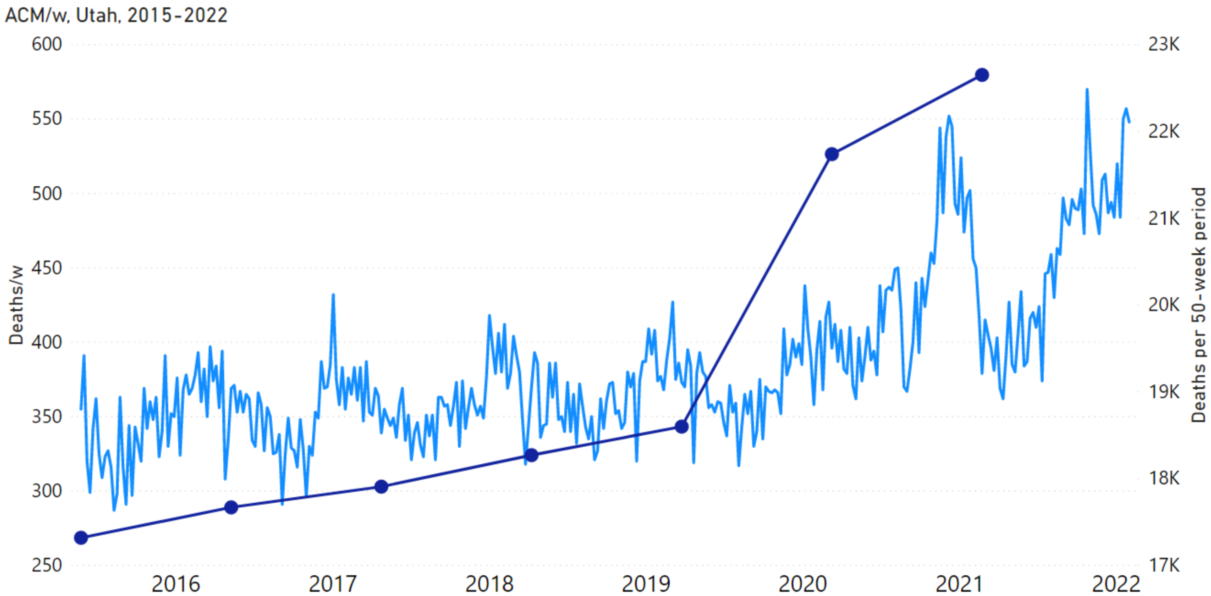
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Tennessee**	**91 144**	**88 685**	**73 230**	**15 455**	**17 914**	**21,10 %**	**24,46 %**
0-24	1 950	1 886	1 808	78	142	4,31 %	7,85 %
25-44	6 538	5 563	4 242	1 321	2 296	31,14 %	54,13 %
45-64	21 321	18 986	16 149	2 837	5 172	17,57 %	32,03 %
65-74	20 633	19 287	15 666	3 621	4 967	23,11 %	31,71 %
75-84	21 700	21 871	17 823	4 048	3 877	22,71 %	21,75 %
85+	19 002	21 092	17 542	3 550	1 460	20,24 %	8,32 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Tennessee**	**179 829**	**144 108**	**140 987**	**35 721**	**38 842**	**24,79 %**	**27,55 %**
0-24	3 836	3 482	3 562	354	274	10,17 %	7,69 %
25-44	12 101	8 228	7 690	3 873	4 411	47,07 %	57,36 %
45-64	40 307	32 050	31 199	8 257	9 108	25,76 %	29,19 %
65-74	39 920	30 640	30 168	9 280	9 752	30,29 %	32,33 %
75-84	43 571	35 212	33 522	8 359	10 049	23,74 %	29,98 %
85+	40 094	34 496	34 846	5 598	5 248	16,23 %	15,06 %



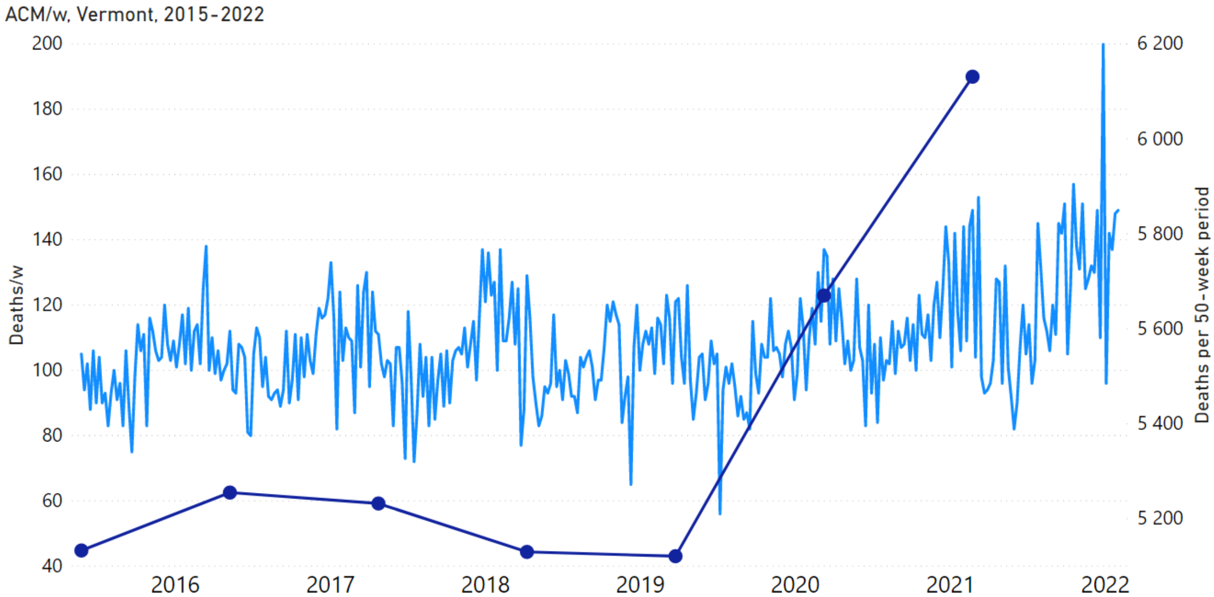
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Texas	**255 483**	**261 182**	**198 282**	**62 900**	**57 201**	**31,72 %**	**28,85 %**
0-24	6 854	6 208	5 914	294	940	4,97 %	15,89 %
25-44	18 140	15 019	11 375	3 644	6 765	32,04 %	59,47 %
45-64	58 218	55 719	41 722	13 997	16 496	33,55 %	39,54 %
65-74	55 639	56 323	40 744	15 579	14 895	38,24 %	36,56 %
75-84	58 693	62 941	47 368	15 573	11 325	32,88 %	23,91 %
85+	57 939	64 972	51 159	13 813	6 780	27,00 %	13,25 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Texas	**516 665**	**391 871**	**385 718**	**124 794**	**130 947**	**31,85 %**	**33,95 %**
0-24	13 062	11 536	11 887	1 526	1 175	13,23 %	9,88 %
25-44	33 159	22 116	21 662	11 043	11 497	49,93 %	53,07 %
45-64	113 937	83 234	83 961	30 703	29 976	36,89 %	35,70 %
65-74	111 962	80 588	76 293	31 374	35 669	38,93 %	46,75 %
75-84	121 634	92 634	89 439	29 000	32 195	31,31 %	36,00 %
85+	122 911	101 763	102 476	21 148	20 435	20,78 %	19,94 %



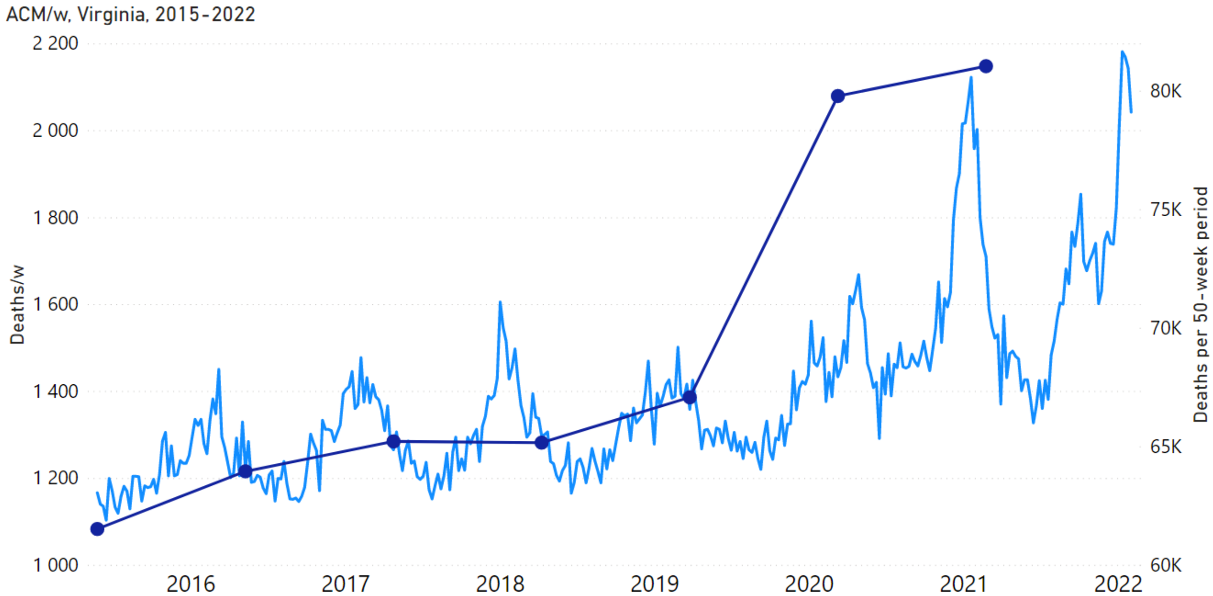
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Utah	**22 658**	**21 739**	**18 599**	**3 140**	**4 059**	**16,88 %**	**21,82 %**
0-24	630	561	657	-96	-27	-14,61 %	-4,11 %
25-44	1 633	1 508	1 209	299	424	24,73 %	35,07 %
45-64	4 290	3 719	3 101	618	1 189	19,93 %	38,34 %
65-74	4 324	4 038	3 293	745	1 031	22,62 %	31,31 %
75-84	5 533	5 427	4 637	790	896	17,04 %	19,32 %
85+	6 248	6 486	5 702	784	546	13,75 %	9,58 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Utah	**44 397**	**36 869**	**35 575**	**7 528**	**8 822**	**20,42 %**	**24,80 %**
0-24	1 191	1 261	1 352	-70	-161	-5,55 %	-11,91 %
25-44	3 141	2 514	2 479	627	662	24,94 %	26,70 %
45-64	8 009	6 324	6 377	1 685	1 632	26,64 %	25,59 %
65-74	8 362	6 504	6 049	1 858	2 313	28,57 %	38,24 %
75-84	10 960	9 112	8 468	1 848	2 492	20,28 %	29,43 %
85+	12 734	11 154	10 850	1 580	1 884	14,17 %	17,36 %



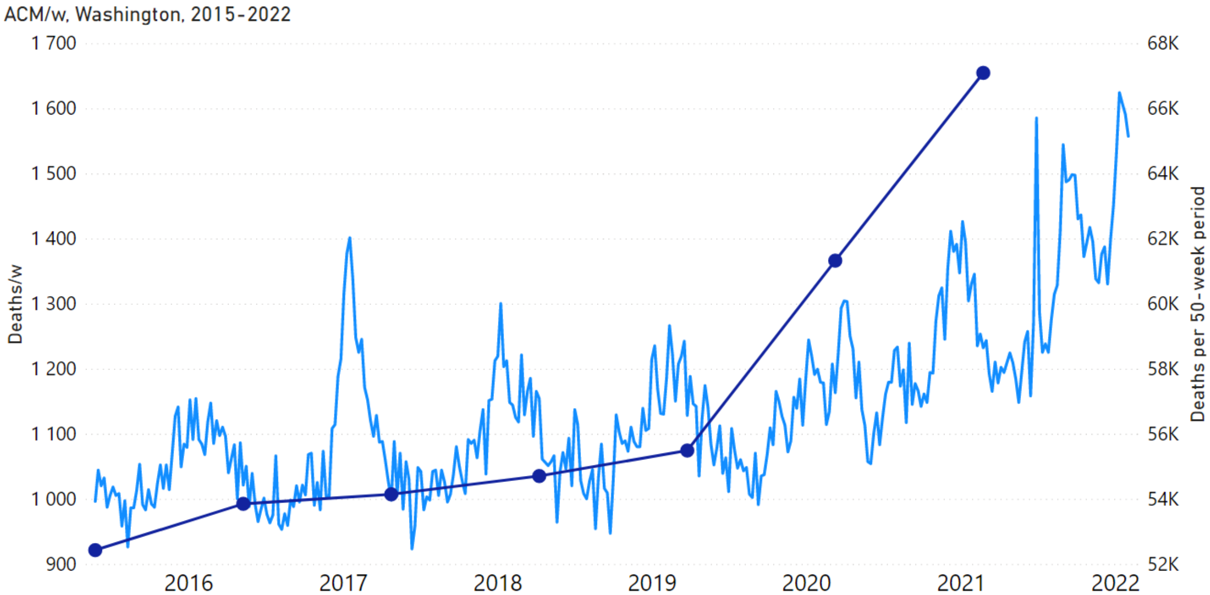
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
Vermont	**6 116**	**5 670**	**5 121**	**549**	**995**	**10,72 %**	**19,43 %**
25-44	65	11		11	65	Infinity	Infinity
45-64	1 066	971	859	112	207	13,04 %	24,10 %
65-74	1 239	1 149	1 041	108	198	10,37 %	19,02 %
75-84	1 584	1 477	1 293	184	291	14,23 %	22,51 %
85+	2 162	2 062	1 928	134	234	6,95 %	12,14 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
Vermont	**11 786**	**10 251**	**10 487**	**1 535**	**1 299**	**14,97 %**	**12,39 %**
25-44	76	22	24	54	52	245,45 %	216,67 %
45-64	2 037	1 717	1 762	320	275	18,64 %	15,61 %
65-74	2 388	2 033	2 013	355	375	17,46 %	18,63 %
75-84	3 061	2 622	2 622	439	439	16,74 %	16,74 %
85+	4 224	3 857	4 066	367	158	9,52 %	3,89 %



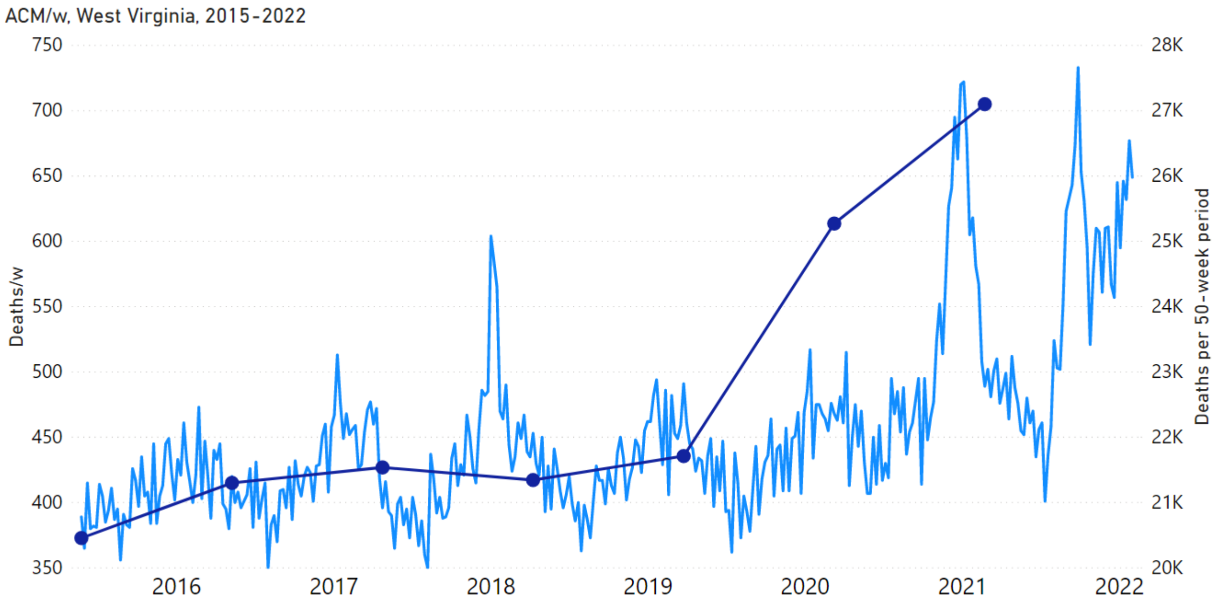
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Virginia**	**81 528**	**79 781**	**67 090**	**12 691**	**14 438**	**18,92 %**	**21,52 %**
0-24	1 629	1 542	1 380	162	249	11,74 %	18,04 %
25-44	4 598	4 014	3 183	831	1 415	26,11 %	44,45 %
45-64	16 023	14 750	12 577	2 173	3 446	17,28 %	27,40 %
65-74	17 048	15 951	13 203	2 748	3 845	20,81 %	29,12 %
75-84	20 275	20 044	16 648	3 396	3 627	20,40 %	21,79 %
85+	21 955	23 480	20 099	3 381	1 856	16,82 %	9,23 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Virginia**	**161 309**	**132 268**	**129 198**	**29 041**	**32 111**	**21,96 %**	**24,85 %**
0-24	3 171	2 763	2 805	408	366	14,77 %	13,05 %
25-44	8 612	6 140	6 011	2 472	2 601	40,26 %	43,27 %
45-64	30 773	25 117	25 368	5 656	5 405	22,52 %	21,31 %
65-74	32 999	25 973	25 049	7 026	7 950	27,05 %	31,74 %
75-84	40 319	32 878	30 842	7 441	9 477	22,63 %	30,73 %
85+	45 435	39 397	39 123	6 038	6 312	15,33 %	16,13 %



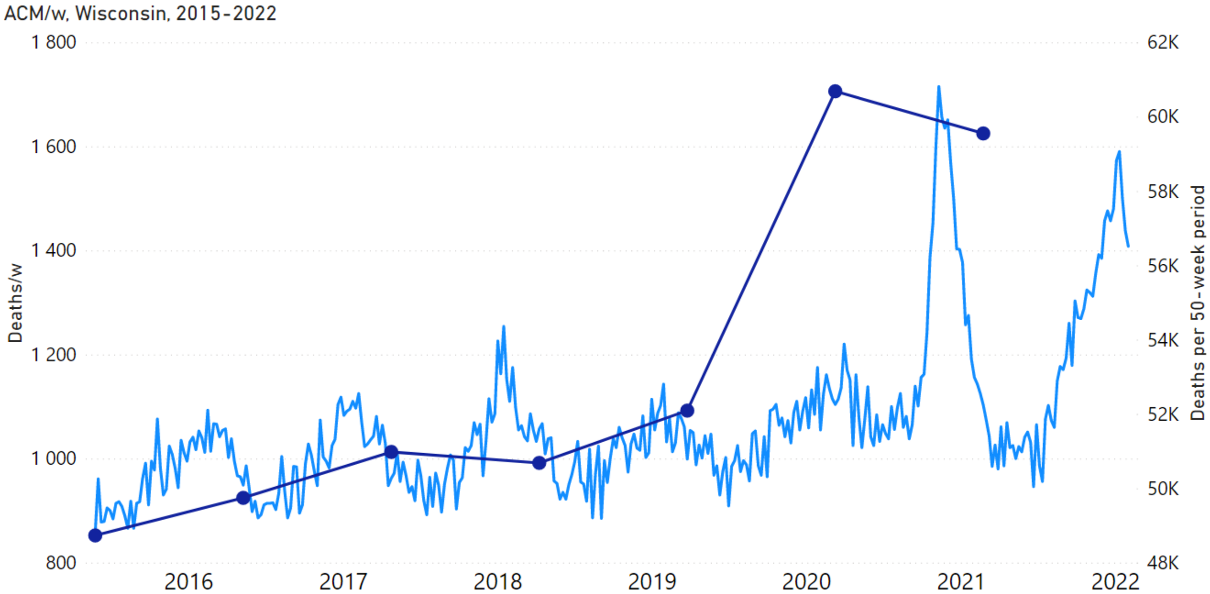
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Washington**	**67 133**	**61 338**	**55 503**	**5 835**	**11 630**	**10,51 %**	**20,95 %**
0-24	1 254	1 151	1 050	101	204	9,62 %	19,43 %
25-44	3 807	3 046	2 547	499	1 260	19,59 %	49,47 %
45-64	12 590	10 944	9 842	1 102	2 748	11,20 %	27,92 %
65-74	14 137	12 474	11 251	1 223	2 886	10,87 %	25,65 %
75-84	16 219	14 840	13 375	1 465	2 844	10,95 %	21,26 %
85+	19 126	18 883	17 438	1 445	1 688	8,29 %	9,68 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Washington**	**128 471**	**110 222**	**108 022**	**18 249**	**20 449**	**16,56 %**	**18,93 %**
0-24	2 405	2 166	2 164	239	241	11,03 %	11,14 %
25-44	6 853	5 007	4 757	1 846	2 096	36,87 %	44,06 %
45-64	23 534	19 810	19 911	3 724	3 623	18,80 %	18,20 %
65-74	26 611	22 149	20 792	4 462	5 819	20,15 %	27,99 %
75-84	31 059	26 296	24 644	4 763	6 415	18,11 %	26,03 %
85+	38 009	34 794	35 754	3 215	2 255	9,24 %	6,31 %



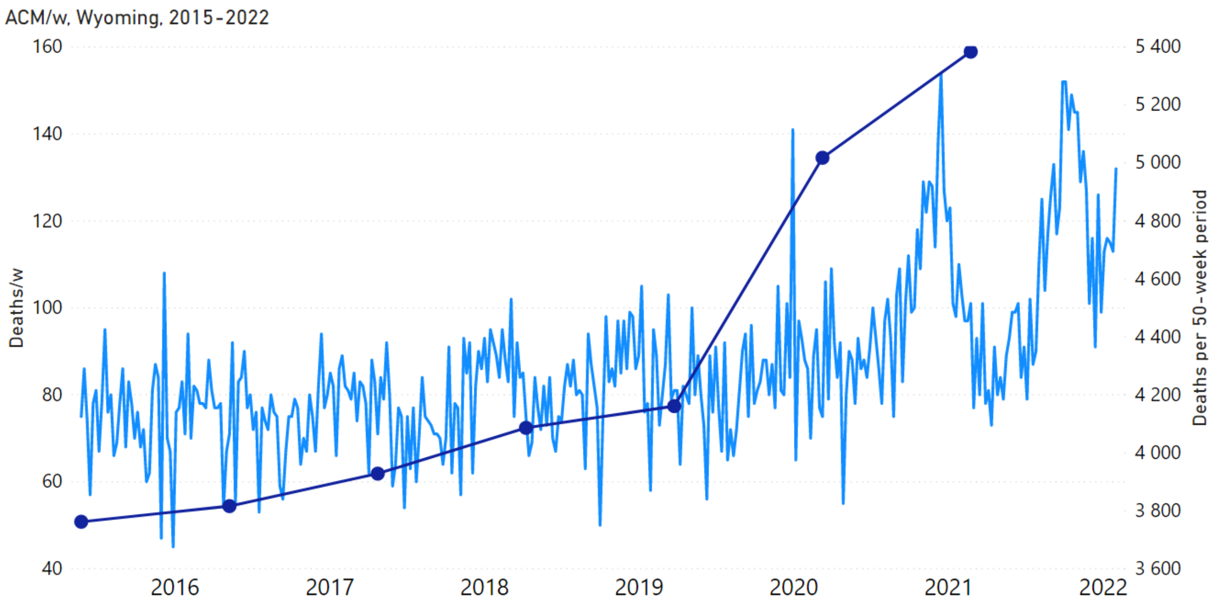
State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
West Virginia	**27 179**	**25 273**	**21 712**	**3 561**	**5 467**	**16,40 %**	**25,18 %**
0-24	74	45	24	21	50	87,50 %	208,33 %
25-44	1 728	1 584	1 284	300	444	23,36 %	34,58 %
45-64	6 056	5 201	4 500	701	1 556	15,58 %	34,58 %
65-74	6 476	5 630	4 782	848	1 694	17,73 %	35,42 %
75-84	6 654	6 503	5 548	955	1 106	17,21 %	19,94 %
85+	6 191	6 310	5 574	736	617	13,20 %	11,07 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
West Virginia	**52 452**	**43 057**	**42 839**	**9 395**	**9 613**	**21,82 %**	**22,44 %**
0-24	119	98	152	21	-33	21,43 %	-21,71 %
25-44	3 312	2 490	2 565	822	747	33,01 %	29,12 %
45-64	11 257	8 956	9 126	2 301	2 131	25,69 %	23,35 %
65-74	12 106	9 325	8 986	2 781	3 120	29,82 %	34,72 %
75-84	13 157	11 029	10 594	2 128	2 563	19,29 %	24,19 %
85+	12 501	11 159	11 416	1 342	1 085	12,03 %	9,50 %



State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Wisconsin**	**59 574**	**60 697**	**52 104**	**8 593**	**7 470**	**16,49 %**	**14,34 %**
0-24	994	989	941	48	53	5,10 %	5,63 %
25-44	3 025	2 629	2 095	534	930	25,49 %	44,39 %
45-64	10 348	9 721	8 558	1 163	1 790	13,59 %	20,92 %
65-74	11 824	11 566	9 558	2 008	2 266	21,01 %	23,71 %
75-84	14 589	14 815	12 685	2 130	1 904	16,79 %	15,01 %
85+	18 794	20 977	18 267	2 710	527	14,84 %	2,88 %

State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Wisconsin**	**120 271**	**102 801**	**100 748**	**17 470**	**19 523**	**16,99 %**	**19,38 %**
0-24	1 983	1 849	2 072	134	-89	7,25 %	-4,30 %
25-44	5 654	4 094	4 093	1 560	1 561	38,10 %	38,14 %
45-64	20 069	17 000	16 852	3 069	3 217	18,05 %	19,09 %
65-74	23 390	18 644	17 492	4 746	5 898	25,46 %	33,72 %
75-84	29 404	24 819	23 580	4 585	5 824	18,47 %	24,70 %
85+	39 771	36 395	36 659	3 376	3 112	9,28 %	8,49 %

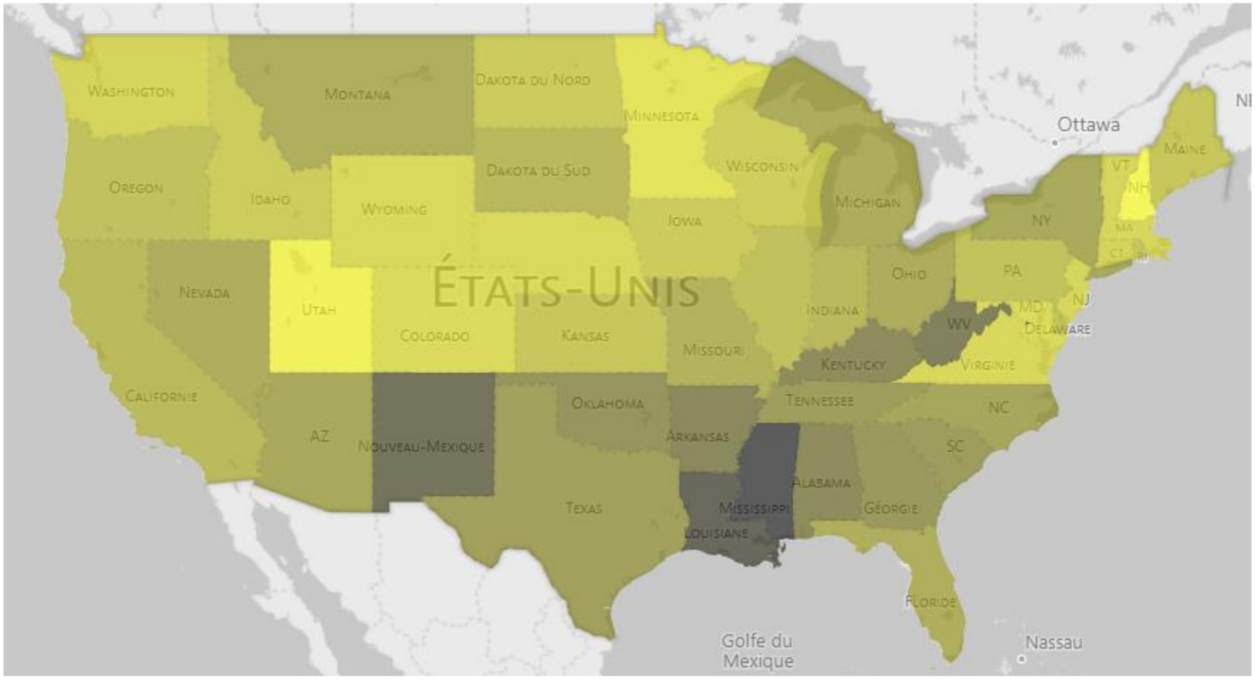


State	w50c	w50c-1	w50c-2	pVax-pCVD	Vax-pCVD	pVax-pCVD/pCVD	Vax-pCVD/pCVD
⊟ **Wyoming**	**5 385**	**5 018**	**4 161**	**857**	**1 224**	**20,60 %**	**29,42 %**
25-44	77	24		24	77	Infinity	Infinity
45-64	1 167	992	868	124	299	14,29 %	34,45 %
65-74	1 308	1 128	883	245	425	27,75 %	48,13 %
75-84	1 352	1 354	1 037	317	315	30,57 %	30,38 %
85+	1 481	1 520	1 373	147	108	10,71 %	7,87 %

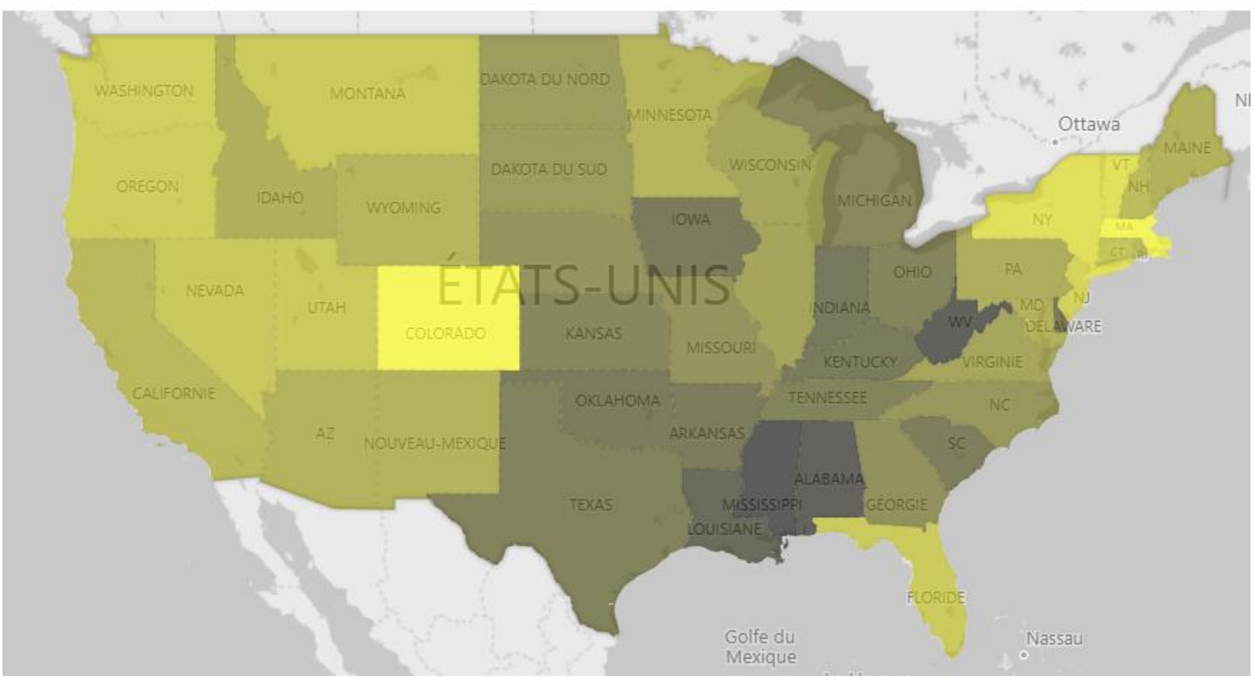
State	w100c	w100c-1	w100c-2	xDc(100)1	xDc(100)2	xDc(100)1%	xDc(100)2%
⊟ **Wyoming**	**10 403**	**8 247**	**7 744**	**2 156**	**2 659**	**26,14 %**	**34,34 %**
25-44	101	23		78	101	339,13 %	Infinity
45-64	2 159	1 662	1 677	497	482	29,90 %	28,74 %
65-74	2 436	1 736	1 606	700	830	40,32 %	51,68 %
75-84	2 706	2 115	1 954	591	752	27,94 %	38,49 %
85+	3 001	2 711	2 507	290	494	10,70 %	19,70 %

Appendix B – Poverty and obesity maps of the USA

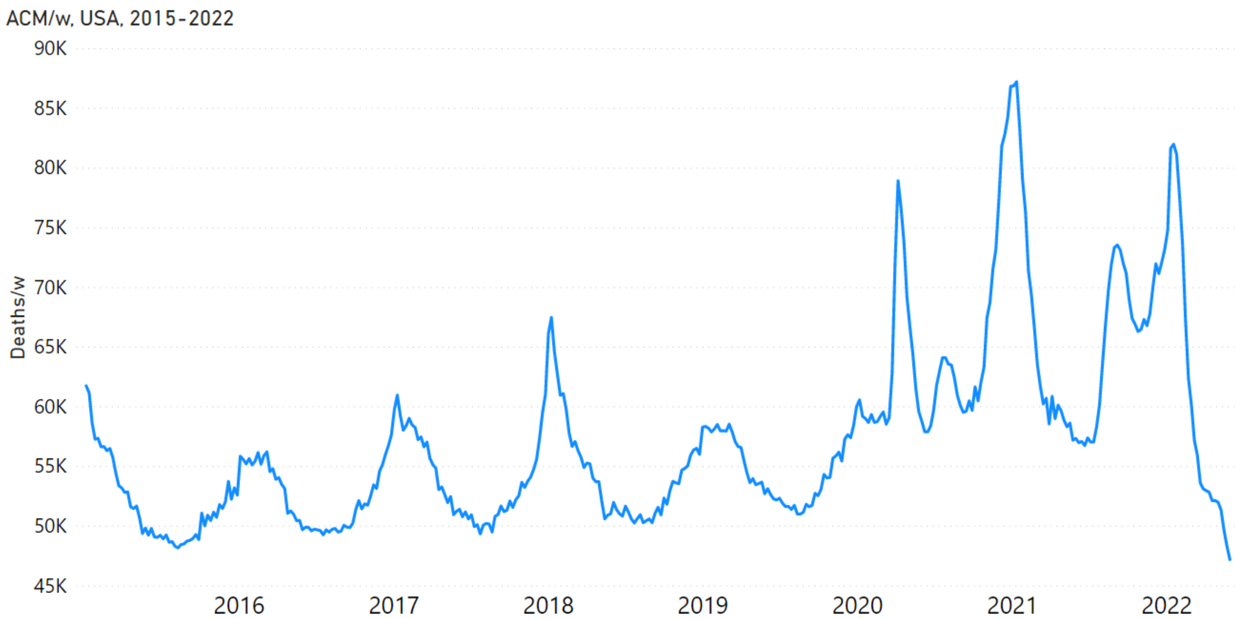
POVERTY IN THE USA



OBESITY IN THE USA



Appendix C – ACM/w in the USA from 2015 to most recent data



Data for this graph were retrieved from the CDC:

https://gis.cdc.gov/grasp/fluview/mortality.html

Evaluating the Effect of Lockdowns On All-Cause Mortality During the COVID Era: Lockdowns Did Not Save Lives

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John Johnson
Harvard University

Evaluating the Effect of Lockdowns On All-Cause Mortality During the COVID Era: Lockdowns Did Not Save Lives

John A. Johnson[1]
Denis Rancourt[2]

Abstract

The USA and its 50 state jurisdictions provide a natural experiment to test whether excess all-cause deaths can be directly attributed to implementing the social and economic structural large-scale changes induced by ordering general-population lockdowns. Ten states had no lockdown impositions and there are 38 pairs of lockdown/non-lockdown states that share a land border. We find that the regulatory imposition and enforcement of state-wide shelter-in-place or stay-at-home orders conclusively correlates with larger health-status-corrected, per capita, all-cause mortality by state. This result is inconsistent with the hypothesis that lockdowns saved lives.

Introduction

On March 11, 2020, the World Health Organization declared a pandemic based on a reported outbreak in Wuhan, China of COVID-19 (hereafter COVID), the respiratory illness purportedly caused by the SARS-CoV-2 virus. On March 13, 2020 a national emergency was declared in the United States concerning the COVID-19 outbreak. In the US, this declaration resulted in a heterogeneous set of responses from health authorities and government officials in various states. Among those varied, state-wise policy responses, most states issued shelter-in-place or stay-at-home orders in March and April of 2020 (hereafter referred to as "lockdowns").

The motivation for these lockdown measures was to slow the spread of COVID-19 by limiting social interactions, under the assumption that the disease spreads by person-to-person contact. However, due to the independence of state governance in the US, the lockdown measures had a wide range of implementation and enforcement, with some states foregoing lockdowns altogether.

These differences in state-wise decisions to either lockdown or not thus establish a useful experiment to test the hypothesis that lockdowns saved lives. This hypothesis predicts that there should have been fewer deaths (per capita) in states that implemented lockdowns, and more deaths in states that did not, after adjusting for differences in the health status of the state populations, if all other factors are presumed to have lesser impact. The data available to test these predictions can be found in all-cause mortality (ACM) by time and by state, reported by the CDC.

[1] Harvard University, 60 Garden St., Cambridge, MA 02138
[2] Ontario Civil Liberties Association (ocla.ca)

As demonstrated by other investigators (for example Rancourt, Baudin & Mercier 2021), ACM sidesteps the difficult issue of cause-of-death assignation, which is political in nature, and as a result susceptible to bias (e.g. Ealy et al. 2020). The correct dominant cause of death is rarely known in the case of respiratory illnesses, and the death is normally not monocausal.

The advantage of analyzing ACM is that deaths in the US are recorded with high fidelity (no reporting bias or underreporting). Once recorded, a death is a death, regardless of how the cause is assigned on the death certificate. If lockdowns are effective in preventing deaths due to the spread of a disease during a pandemic, then regions that implemented lockdowns should have experienced fewer per capita deaths from all causes, if there are no overriding confounding factors.

Data and Methodology

Our goal is to assess the efficacy of lockdowns in saving lives during the COVID era by comparing the total number of deaths from all causes in pairs of states: one state with a lockdown, and a state without a lockdown that shares a border with the lockdown state. We also examined the lockdown states that do not share a border with any non-lockdown state, for completeness.

We identified non-lockdown states by examining administrative and executive orders issued during March-April 2020 by state governments in response to the pandemic declarations of the WHO and of federal and state governments. Most of these orders have been archived at the website Ballotpedia.com, and we located the orders for which the links were no longer valid by searching state government websites. A full list of the URLs for these orders are listed in Table # in the Appendix. We assigned a “stringency” score to each executive order based on the language of the lockdown order for the citizens of the state:

Ordered/mandated: 3
Directed: 2
Suggested/encouraged: 1
No order: 0

We found that there were seven (7) states that had scores of 0 because they did not issue stay-at-home orders: North Dakota, South Dakota, Wyoming, Iowa, Oklahoma, Nebraska, and Arkansas. There were an additional three (3) states that had scores of 1 because the governments only suggested or encouraged citizens to stay at home, but did not require them to do so, nor provided means of enforcement: Utah, Kentucky and Tennessee.

Our criterion for lockdown versus non-lockdown states differs from previous studies in its simplicity (i.e. focusing only on the stringency of the language in the executive orders). But our resulting list of non-lockdown states includes all seven states listed as non-lockdown on

Ballotpedia, and includes all four non-lockdown states identified by the CDC-sponsored study of Moreland et al. (2020).

We compared the outcomes of these ten non-lockdown states with lockdown states that share a border, under the assumption that viral spread is not impeded by state boundaries. In this study we focus on the total all-cause mortality (ACM) over a specified time period as the metric of lockdown efficacy. We use three time periods as described below.

We downloaded comma-separated-value (csv) files containing ACM per week for each state from the CDC Wonder website. The CDC ACM data are listed by “season,” and these seasons span calendar years. We remapped the season weeks onto a time axis in units of fractional years. For example, for the 2013-2014 season, weeks 1-39 correspond to 2014, while week numbers greater than 39 occur in 2013.

We divided the weekly ACM data for each state by that state’s population (US Census, April 1, 2020), resulting in the number of deaths per capita, per week (D_{pcw}). Throughout this report we express D_{pcw} as the number of deaths per 10,000 residents.

An additional correction step is necessary to allow for accurate state-by-state comparisons of mortality. Differences in age distributions, obesity rates, poverty levels, physical and mental disability rates, and other health determinants will lead to intrinsic differences in D_{pcw} in various states. These differences collectively manifest in an offset in D_{pcw} seen during non-pandemic years (prior to 2020).

For example, Figure 1 shows a comparison of the D_{pcw} between New York and Florida during the years 2014-2020. As with all state-wise comparisons, New York and Florida have remarkably similar temporal variations in D_{pcw} from week to week and from year to year, yet also have a clear and nearly constant offset.

We correct for this offset by computing a factor H_{state}, which is the median value of the ratio of a state’s D_{pcw} and the D_{pcw} of a reference state from January 1, 2014 through December 31, 2020. We chose New York as a reference state for computing H_{state}. This choice of reference state is arbitrary, but the large population of New York means that, in most cases, the error in H_{state} is dominated by Poisson errors in the D_{pcw} of the state of interest.

In the example shown in Figure 1, the health-status correction factor of Florida is $H_{state} = 0.537$, indicating that New York experienced 53.7% fewer D_{pcw} than Florida in the years 2014 to 2020, likely owing in part to the older population in Florida. For each state-wise comparison of D_{pcw} we adopt this ratio as a correction factor to bring the pair of states onto the same scale, allowing for a health-status-corrected comparison of mortality during the pandemic period.

This health-status correction factor is justified since we are performing a differential comparison between states with and without lockdowns. We are asking, “Following the enactment of lockdown measures, what is the fractional difference between the adjusted per capita ACM in

each pair of states?" This assumes that after removing differences in the health status of the populations of neighboring states, the largest effect on the adjusted per capita ACM was the enactment of a lockdown. This assumption is justified given that the lockdowns are expected to result in massive disruptions to national and regional economies, healthcare systems, and general social fabric.

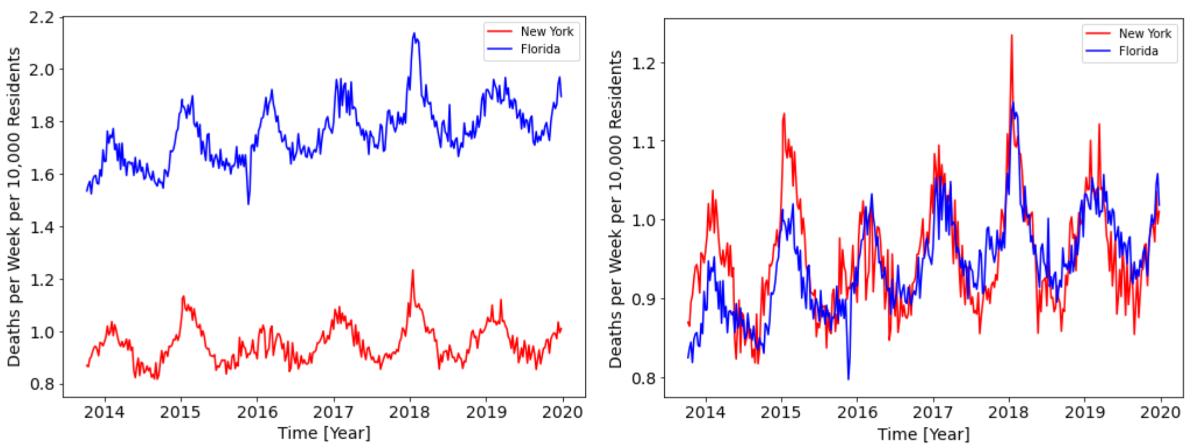


Figure 1: *The deaths per capita, per week (D_{pcw}) in Florida (red) and New York (blue). The left-hand panel shows the offset in D_{pcw}, which we attribute to differences in each state's population health status (age structure, poverty level, obesity rate, etc.). The panel on the right shows the corrected D_{pcw}, which allows for a differential comparison between these two states from 2020 onward.*

To quantify the effect of lockdowns on mortality during the COVID period we calculate the integrated (total) health-status-corrected deaths per capita, D_{tot}, over a chosen time period. We then compute the ratio of D_{tot} for each pair of states, denoted by R (lockdown divided by non-lockdown). We use three different time periods over which we expect D_{tot}, and R, to capture the effects of the lockdown measures:

$D_{tot,1}$: Sum over lockdown period of the lockdown state.
$D_{tot,2}$: Sum over the period of "COVID peak 1" (cp1) as identified by Rancourt et al. (2021; week 11 through week 25 of 2020)
$D_{tot,3}$: Sum over entire period from March 11, 2020 to December 31, 2021

We estimate the uncertainties of these ratios by propagating Poisson errors in each total (uncorrected) ACM over each time period. Thus, these estimated uncertainties are the calculated errors arising from counting statistics. We expect that there are no other significant sources of error, given the integrity of the ACM data for the United States.

Throughout this paper we report the 95% confidence intervals for our integrated, population-normalized and health-status-corrected mortality ratios for each pair-wise

comparison of lockdown and non-lockdown states, and for the health-status-corrected integrated per capita mortalities that we report.

Figure 2 illustrates the results of our ACM analysis in three panels for the example of Louisiana (lockdown state) compared to Arkansas (non-lockdown state). The date of the WHO pandemic announcement is shown in all panels with a vertical dashed line.

The first panel shows the health-status-corrected ACM per week, per 10,000 residents for Louisiana. The “COVID peak 1” (cp1) 15-week time period is shown as a blue shaded region, and for Louisiana covers a significant spike in the ACM, which also appears in the ACM per week of the entire US (Rancourt et al., 2021). The lockdown period for Louisiana is shown as a pink shaded region, and for this example the lockdown period is somewhat shorter than the cp1 time span.

The second panel shows the health-status-corrected ACM per week, per 10,000 residents for Arkansas (blue line), which we designate as a non-lockdown state. For ease of comparison, the ACM per week, per capita for Louisiana is also shown (gray line).

The final panel shows the weekly difference of the health-status-corrected per capita ACM in the lockdown state minus the health-status-corrected per capita ACM in the non-lockdown state.

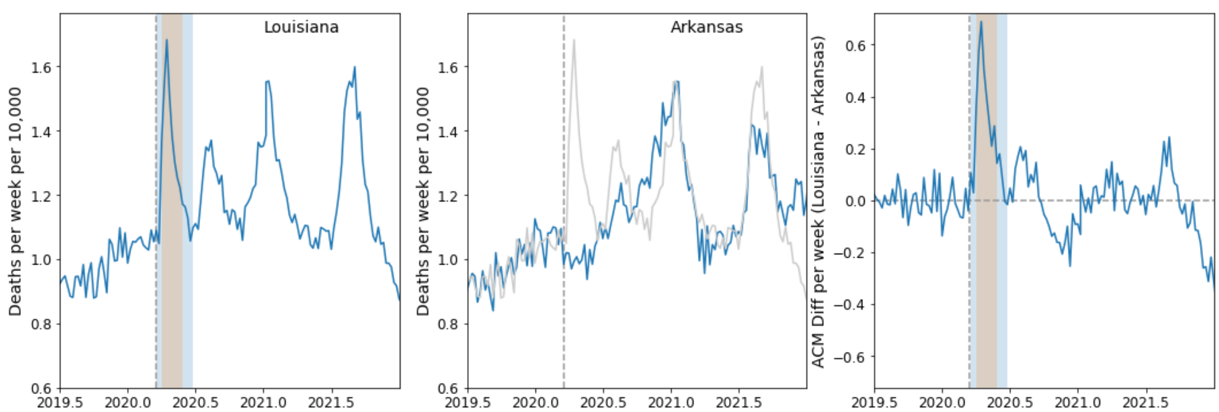


Figure 2: Left - *The deaths per week in Louisiana, a lockdown state, corrected for the difference in population health status between Louisiana and Arkansas (blue line). The dashed line shows the date of the WHO pandemic announcement. The blue shaded region corresponds to the “COVID peak 1” (cp1) time period. The orange shaded region shows the time period of Louisiana’s lockdown.* Middle - *The same as the left panel, but for the non-lockdown state, Arkansas. The gray line is the deaths per week for Louisiana, for ease of comparison.* Right - *The weekly differences of the deaths per week in Louisiana as compared to Arkansas. The shaded regions are the same as in the left-hand panel.*

Results

Our results are summarized in the figures below.

In Figures 3, 4, and 5, the y-axis lists all 38 lockdown/non-lockdown pairs of states used for comparing mortality outcomes, with the lockdown state listed first, followed by the non-lockdown state. The blue dots show the point-estimate of the ratio, R, and the associated error bars show the 95% confidence interval; the vertical dashed line marks unity. Values to the left of the vertical line indicate instances in which the lockdown state experienced fewer health-status-corrected per capita deaths than the non-lockdown state. Values to the right of the line indicate that the lockdown state experienced more health-status-corrected per capita deaths than the non-lockdown state.

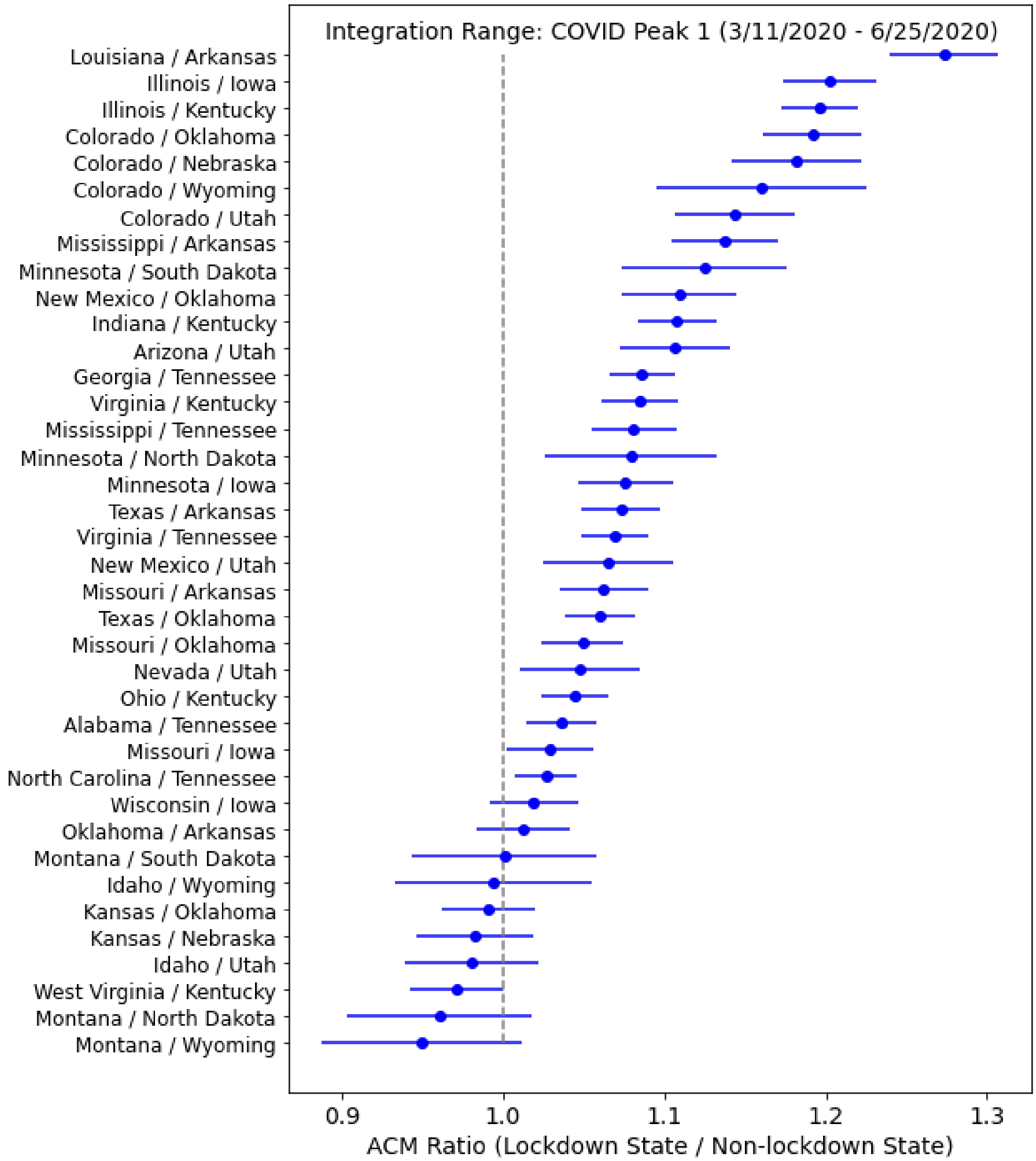


Figure 3: *The health-status adjusted per capita ACM ratio (R) for each neighboring pair of states listed on the y-axis. The ratio is based on summing all deaths in each state over the time period corresponding to the COVID peak (3/11/2020 - 6/24/2020). The error bars show the 95% confidence interval for each pair's ratio. Ratios to the left of the vertical line indicate that fewer deaths occurred in the lockdown state than in the non-lockdown state, while ratios to the right of the vertical line indicate that states with lockdowns experienced more deaths.*

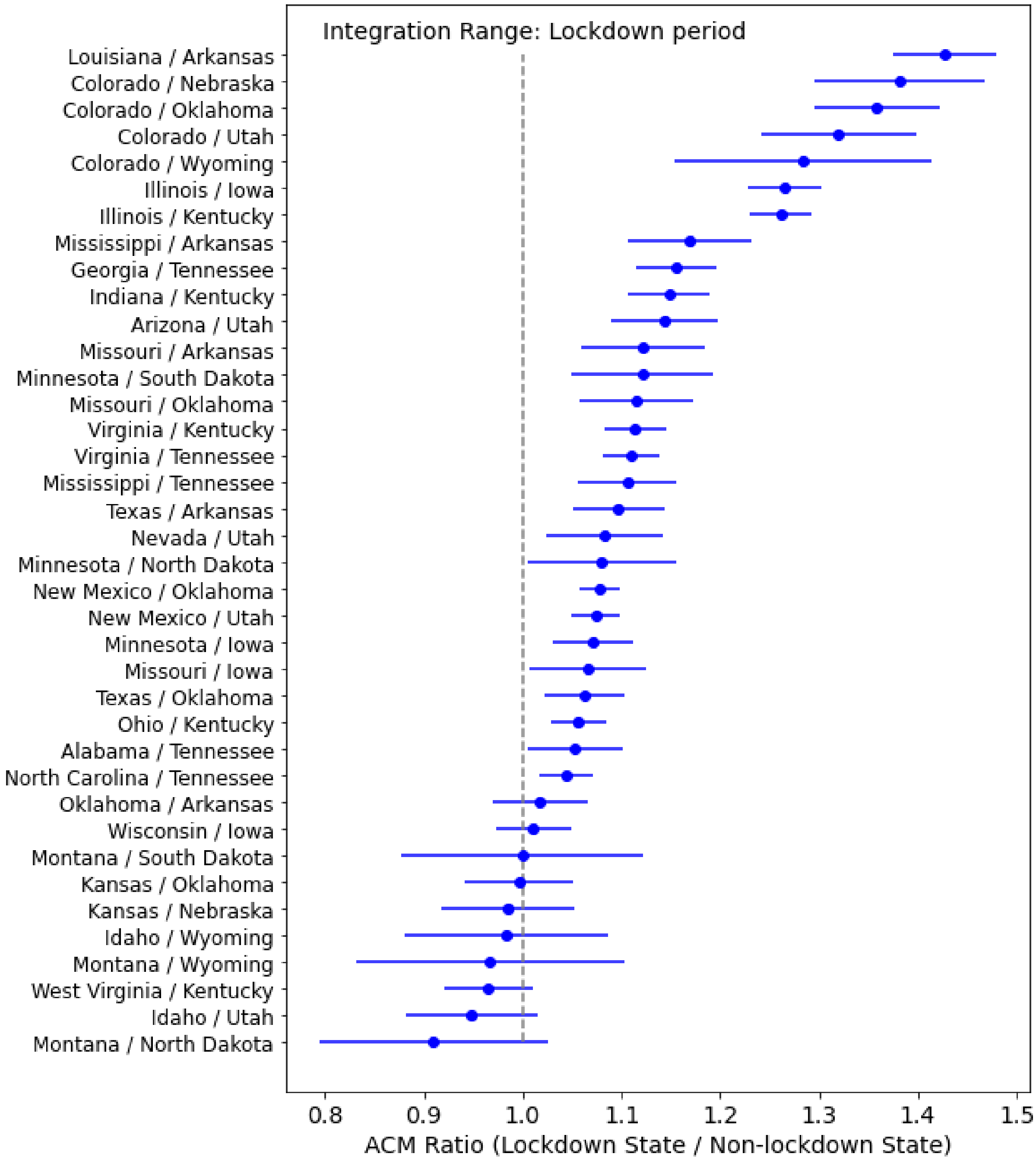


Figure 4: *The health-status adjusted per capita ACM ratio (R) for each neighboring pair of states listed on the y-axis. The ratio is based on summing all deaths in each state over the time period corresponding to the lockdown state's lockdown duration. The error bars show the 95% confidence interval for each pair's ratio. Ratios to the left of the vertical line indicate that fewer deaths occurred in the lockdown state than in the non-lockdown state, while ratios to the right of the vertical line indicate that states with lockdowns experienced more deaths.*

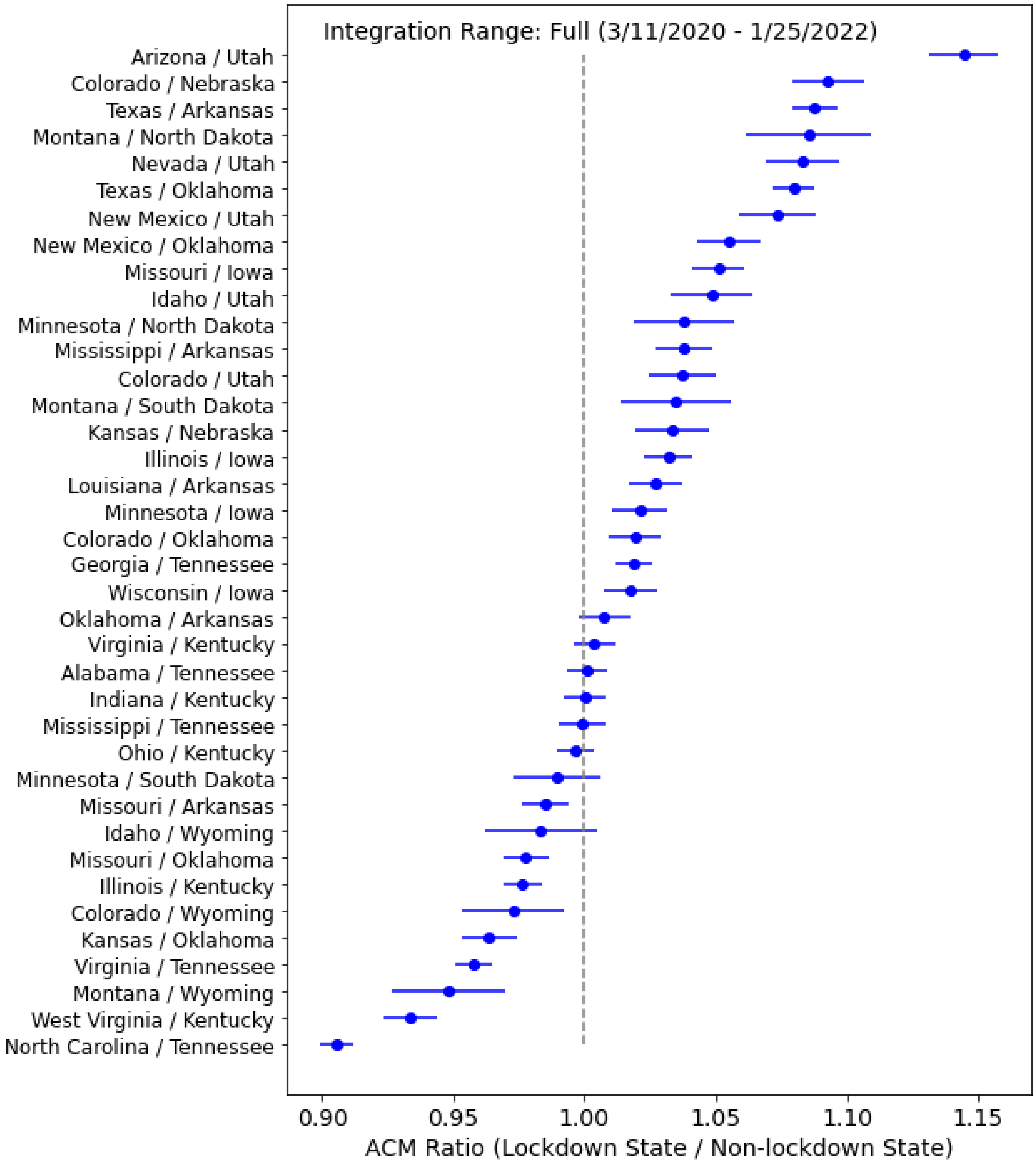


Figure 5: *The health-status adjusted per capita ACM ratio (R) for each neighboring pair of states listed on the y-axis. The ratio is based on summing all deaths in each state over the full "COVID Era" in our data set (March 11, 2020 - Jan 25, 2022). The error bars show the 95% confidence interval for each pair's ratio. Ratios to the left of the vertical line indicate that fewer deaths occurred in the lockdown state than in the non-lockdown state, while ratios to the right of the vertical line indicate that states with lockdowns experienced more deaths.*

If lockdowns saved lives, then we would expect that most of the ACM ratios (R) would be less than one. Instead, we see the opposite. For all three integration periods, the majority of ratios are larger than one. For the cp1 (lockdown, full) period, 28 (28, 21) pairs have ACM ratios (R) larger than one, while 0 (0, 9) pairs have ratios less than one, and the remaining 10 (10, 8) pairs have R indistinguishable from unity at 95% confidence.

Thus, our analysis of R values for three time periods during which lockdowns are expected to have an effect shows that the ACM data from the past two years is inconsistent with the hypothesis that lockdowns saved lives. On the other hand, our results are consistent with the conclusion of Rancourt et al. (2021) that the excess deaths in the COVID period in the USA are caused by the government and medical measures, and responses to the declared pandemic.

Figure 6 shows the health-status-corrected integrated deaths per capita for the 15-week “COVID peak 1” period (cp1; weeks 11 through 25 of 2020) for all the states individually (red) and for the same 15-week integration window in 2019 (blue) and 2018 (green). Here, the states are ordered, top to bottom, in decreasing order of average state-wise population density, which is often presumed to be a factor in the spread of a contagious disease. The state names in magenta correspond to our ten non-lockdown states having lockdown stringency scores of 0 or 1. The state names in cyan are the lockdown states that share a border with a non-lockdown states, which we used in our calculation of R.

The values of health-status-corrected integrated all-cause mortality in the 15-week “cp1” periods of 2019 and 2018 are tightly constrained for all states to a value of approximately 14 deaths per 10,000 (Figure 6), whereas the corresponding values in the COVID period are widely different from state to state, ranging from the 2019 baseline value to as high as 25 per 10,000 for New Jersey, and being typically as large as 15 to 21 per 10,000. Non-lockdown states have names on the y-axis colored magenta, while the lockdown states used as our comparands in calculating R are colored cyan.

Figure 6 shows that most of our ten non-lockdown states have health-status-corrected integrated all-cause mortality in the 15-week cp1 on the pre-COVID (2018 and 2019) baseline value of approximately 14 per 10,000, whereas most of the states with lockdown stringency scores of 2 and 3 have mortality rates well above the pre-COVID baseline values.

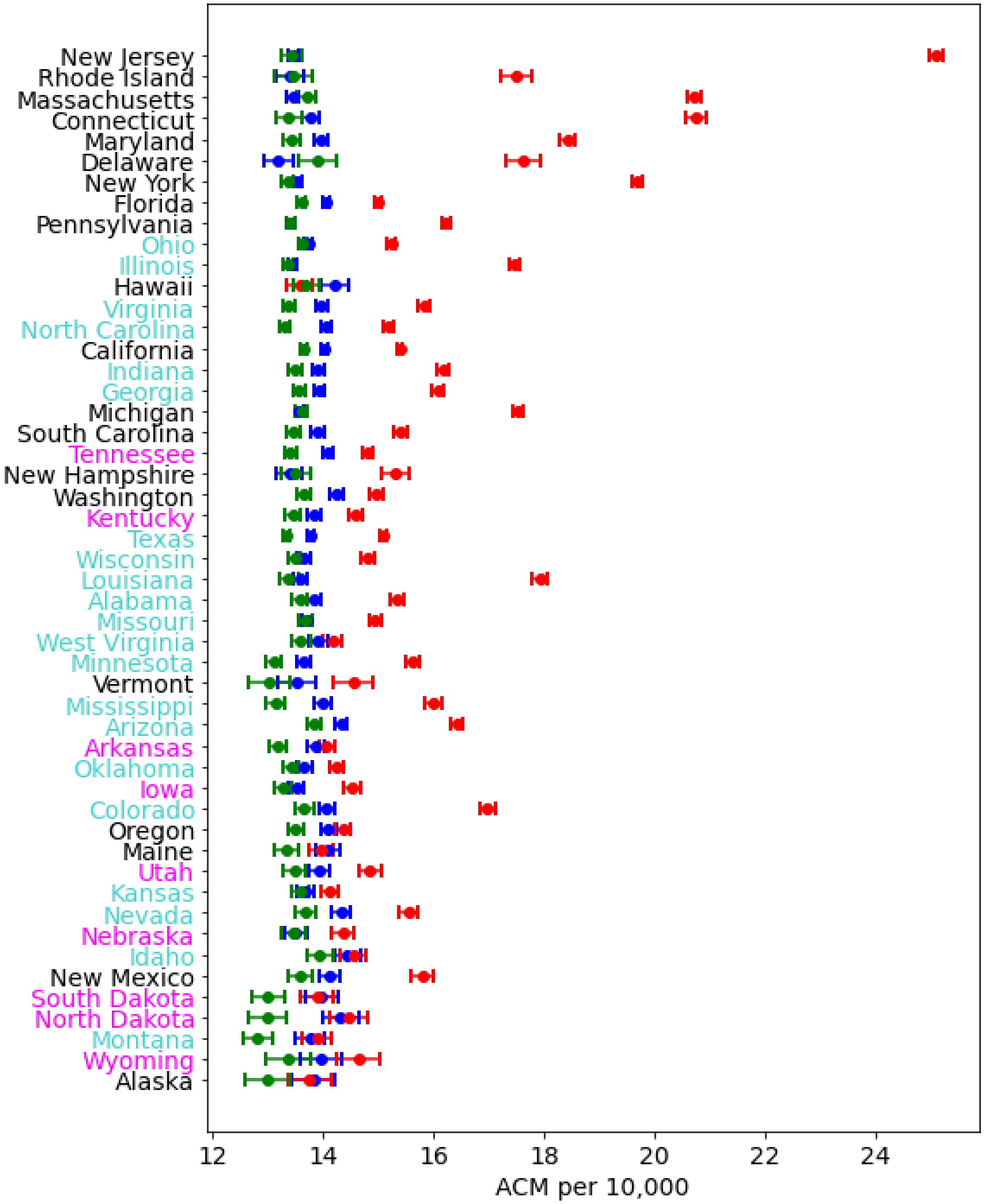


Figure 6: *Integrated health-status corrected ACM over the cp1 period (March 11-June 29 2020; red) compared to the same time period in 2019 (blue) and 2018 (green). States ordered from top to bottom in decreasing population density. Magenta indicates non-lockdown states while cyan denotes lockdown states that share a border with non-lockdown states.*

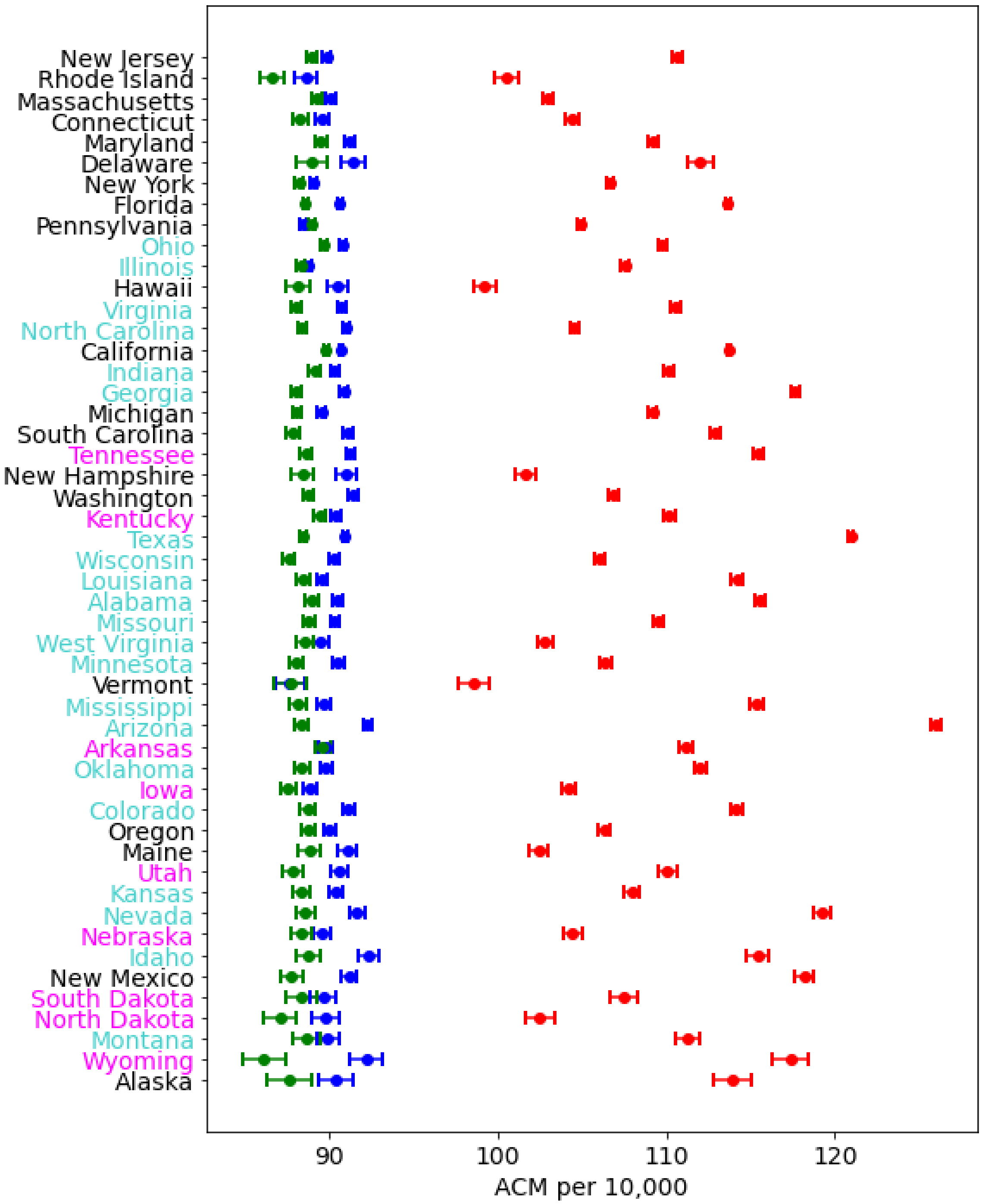


Figure 7: *Integrated health-status corrected ACM over the full COVID period (March 11, 2020 - December 31, 2021; red) compared to the same time period starting in 2018 (blue) and 2016 (green). States ordered from top to bottom in decreasing population density. Magenta indicates non-lockdown states while cyan denotes lockdown states that share a border with non-lockdown states.*

While a precise estimation of the excess mortality due to lockdowns is beyond the scope of this paper, we can make a rough estimate based on Figure 6. The three most populous states (California, Texas, Florida) have above-baseline COVID-period increases of approximately 1 per 10,000. On the basis of one calendar year (52 weeks), and for a population equal to that of the entire USA, this would correspond to approximately 110,000 deaths, which could be attributed directly to the impacts of ordering lockdowns and which would not have occurred if lockdowns had not been implemented. This value is consistent with the lockdown excess mortality estimate of 97K/year by Mulligan & Arnot (2022).

Figure 7 shows a calculation analogous to that employed in Figure 6, but for the entire COVID period covered by our dataset (March 11, 2020 through December 31, 2021), rather than solely for the time-window of cp1. On this time-integration period, we no longer distinguish a state-wise differential effect from the lockdowns, which were relatively short and occured at the beginning of the COVID period. What is apparent, instead, is the large all-cause mortality in all states in the COVID period, and the large state-to-state differences in integrated COVID-period mortality. It appears that the specific state-wise lockdowns considered in this article, while having large and well-recognized effects at times near the lockdown periods, have relatively small or undiscerned effects compared to all the COVID-period and state-wise heterogeneous changes that caused excess deaths on the entire COVID period.

The largest modification we made to the ACM data is the health-status correction factor, H_{state}. For each comparison pair we examined ACM ratio, R, and H_{state}. As shown in Figure 8, there is no correlation between R and H_{state} for both the lockdown and non-lockdown state in each comparison pair.

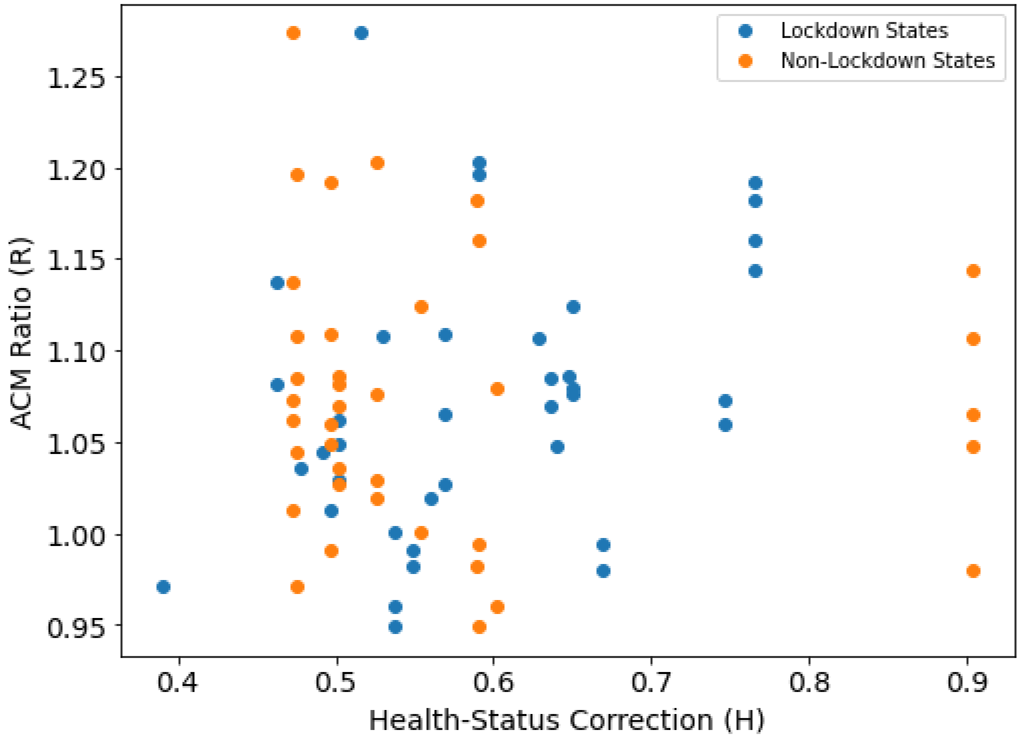


Figure 8: *Integrated health-status corrected ACM ratio (R) versus health-status correction factor H for the lockdown state (blue) and non-lockdown state (orange) in each comparison pair.*

Discussion and Conclusion

The use of lockdowns to “quarantine” the general population of the United States in order to control the spread of an infectious disease is without precedent in the nation’s history. During previous pandemics, only the sick and infirm were quarantined while the rest of the population continued more or less as normal.

This “targeted protection” approach was recommended by medical professionals in the Great Barrington Declaration in 2020, demonstrating that alternatives to lockdowns existed and were well understood within the medical community. As recently as 2019 the World Health Organization advocated a similar approach in its recommendations for mitigating the risks of an influenza pandemic while making no mention of lockdown measures for the general population (WHO 2019). Indeed, the WHO report specifically states that quarantining exposed individuals is “not recommended because there is no obvious rationale for this measure” (see their Tables 1 and 4). Similarly, the *Influenza pandemic preparedness action plan for the United States* makes no mention of lockdowns and states that “...classical measures designed to reduce the risk of introduction and transmission of some infectious agents, such as clinical screening and quarantine at ports of entry, are not likely to be effective” (Strikas et al. 2002).

In their review of the available literature on influenza pandemic interventions, Inglesby et al. (2006) explicitly recommend against quarantine measures in the event of an influenza pandemic, for both sick and healthy individuals, because the societal costs are expected to far outweigh the benefits. They concluded, “[E]xperience has shown that communities faced with epidemics or other adverse events respond best and with the least anxiety when the normal social functioning of the community is least disrupted.” These recommendations extend beyond preparing for and responding to influenza pandemics. In a report titled *Preparedness for a High-Impact Respiratory Pathogen Pandemic*, the authors conclude that quarantine is among the least effective non-pharmaceutical measures in containing disease spread (Johns Hopkins Center for Health Security 2019).

Thus, the lockdown measures implemented in 2020 by the majority of US states, as well as many countries worldwide, represented an unprecedented, large-scale experiment in infectious disease control. The all-cause mortality data we have analyzed allows us to test the hypothesis that lockdowns saved lives during the COVID pandemic. We find that these data are inconsistent with this hypothesis; states with lockdowns experienced more all-cause deaths than neighboring states without lockdowns. We therefore conclude that this experiment was a failure of public health policy and that lockdown measures should not be used during future disease outbreaks.

Our finding that all-cause mortality increased in states with lockdowns is consistent with the conclusions of Agrawal et al. (2021) who found statistically significant increases in excess mortality due to shelter-in-place orders in the US and in 43 countries. Similarly, Mulligan & Arnot (2022) estimate that there were 97K/year excess deaths due to lockdowns, with excess mortality distributed equally among all adult age groups, unlike COVID deaths which were most commonly attributed among the elderly.

Given the strong association between general-population lockdown impositions and increased all-cause mortality, demonstrated above (Figures 3-6), it is appropriate to venture hypotheses for the cause or causes of this association.

Obviously, privileged Americans, from the upper-middle and professional classes, did not die from staying at home. However, it is not unreasonable to postulate that the general-population lockdown regulations and orders are nonetheless proxies or statutory indicators of the degree of aggressiveness (including abandonment) with which the societal institutions in the state responded or reacted to the announced pandemic. These institutions would include schools, care homes, hospitals, clinics, disability services, day care facilities, police services, family and social services, and so on.

We tentatively advance this because it is entirely likely that the excess deaths associated with lockdowns are from pools of individuals at particularly high risk of suffering fatal consequences from large and negative disruptions in their lives and support networks. This will be true irrespective of the actual mechanistic cause of death, given the known association between both experienced stress and social isolation and disease severity and mortality, via the impact

on the immune system (Ader and Cohen 1993; Cohen et al. 1991; Cohen et al. 1997; Cohen et al. 2007; Sapolsky 2005; Prenderville et al., 2015; Dhabhar 2014; Rancourt et al. 2021). Indeed, there is ample evidence that the lockdowns are associated with large increases in unemployment and a general worsening of mental health (e.g. Jewell et al. 2020, Czeisler et al. 2020).

The ACM data available through the CDC Wonder website is not disaggregated by both state and demographics, so we were not able to examine which demographic groups were dying, and how they were dying, in each state. However, demographic information is available on a national level, and Mulligan & Arnot (2022) found large increases in excess mortality among people ages 18-65 years which is a demographic that was not at high risk from COVID.

Similarly, Rancourt et al. (2021) found that the temporal and spatial distribution of all-cause mortality in the pandemic period is inconsistent with the effects of a viral respiratory disease. They found evidence that many excess deaths during the pandemic were misdiagnosed bacterial pneumonia infections, likely exacerbated by disruptions to the US healthcare system. Thus, there exists strong evidence supporting the hypothesis that lockdowns placed a sudden and severe stress burden on vulnerable demographics in the US, leading to significant increases in death in those states that used lockdowns as disease control measures.

Nature of the toxicity of the COVID-19 vaccines in the USA

Joseph Hickey*, PhD, and Denis G. Rancourt**, PhD

Ontario Civil Liberties Association, Ottawa, Ontario, Canada

*joseph.hickey@ocla.ca, **denis.rancourt@alumni.utoronto.ca

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Ontario Civil Liberties Association
603-170 Laurier Avenue West
Ottawa, Ontario
Canada K1P 5V5
http://ocla.ca

In this study of the Vaccine Adverse Event Reporting System data (VAERS data, USA) for COVID-19 vaccines we examine the broad features of the data, resolved by:

- major adverse effect (AE) category (death, life-threatening reaction, hospitalization, disability, and all categories),
- vaccine manufacturer (Janssen, Moderna, Pfizer),
- type of injection (shot number in primary series, booster),
- date of injection,
- date of onset or finality of AE, and
- age of the person suffering the AE;

compared to the dates of administration of all the injections, for the different manufacturers and types of injections (see Figure S1), and compared to population characteristics (age structure, poverty, life expectancy, obesity).

We elucidate fundamental aspects of the body's response to these kinds of pulses of toxic charges, related to age-dependent immune efficiency and age-dependent spread of vulnerability, and we identify exponential time decay components in the induced mortalities, with half-life values in the range 13-30 days, possibly arising from the spike protein.

A next version of this report will contain more content, detail, and supplementary materials. Supporting figures illustrating the data and analyses are provided at the end of this report.

We make the following observations and conclusions.

→ The priority targeting of the population “most at risk” at the start of the COVID-19 vaccination campaign had disastrous consequences for that population, with disproportionately large vaccine-induced mortality and AEs (Figure S2).

→ Graphs of AE frequency versus time of onset or finality of the AE in days since injection all show the same time structure (for all resolved AEs and resolved injection characteristics):

- a large initial peak in the first 5 days or less, which is larger and sharper for the mRNA multi-dose injections (Moderna, Pfizer) compared to the virus-vector single-dose injection (Janssen),
- an exponential decay, from ~5 days to ~60 days, with a fitted half-life decay time typically falling in the range 13-30 days, with this same behaviour occurring for all three manufacturers and for all the main categories of AEs, and
- a plateau or “second wave” of AEs at long times, beyond ~60 days and up to ~350 days since injection, which largely consists of AEs having associations with COVID-19 itself. (Figures S3 through S5)

→ Furthermore, the large initial peak in the first 5 days or less ($x < 5$ days) is significantly smaller for a first dose than for a second or third dose, for both Pfizer and Moderna, while the half-life for the exponential part (5 days $\leq x <$ 60 days) is concomitantly larger for the later doses (Figure S5).

→ The observed exponential decay implies a causal link between death (or AE) and injection, up to ~60 days. Accidental deaths would have a uniform (constant) distribution versus time since injection (versus “x”), mathematically corresponding to an infinite decay time.

→ It is reasonable to postulate that the 13-30 day half-life corresponds to the half-life in the body of a toxic component present in or produced by the vaccines, such as the spike protein; and that the initial peak (< 5 days) is due to a toxic component or adjuvant mostly present in the mRNA injections, such as the cationic lipids.

→ It is also reasonable to postulate that there is an enhanced immune response against the vaccine component that causes the initial (x < 5 days) peak of deaths, in the later doses compared to a first dose (Figure S5). If the initial immune response partially debilitates mRNA delivery to cells and organs in the body, then spike-protein cumulative toxicity leading to death could be delayed, with relatively less deaths in the exponential decay phase (5 days ≤ x < 60 days) and longer decay half-lives, for doses in addition to a first dose, as observed (Figure S5).

→ Thus, it would appear that the enhanced initial (< 5 days) immune response partially disables spike protein production and spread, which, in theory, would make the vaccine both less toxic and less effective (if it ever is effective) in doses and boosters beyond

the first dose. In fact, we do observe reductions of overall toxicity with increasing doses and boosters, as per Table 1.

	Pfizer	Moderna	Janssen
first	8.08 (0.48)	15.08 (0.82)	20.4 (2.2)
second	5.76 (0.44)	10.37 (0.75)	-
primary	7.03 (0.33)	12.96 (0.56)	20.4 (2.2)
booster	3.20 (0.58)	3.18 (0.66)	3.8 (3.8)
all	7.77 (0.32)	13.38 (0.53)	26.7 (2.5)
12 to 17	0.60 (0.42)	-	-
18 to 64	2.64 (0.37)	3.47 (0.52)	10.6 (1.7)
65 plus	19.7 (1.9)	25.5 (2.1)	79. (12.)

Table 1. Total number of VAERS deaths divided by total number of doses delivered in the same period (2021) to the same group (all values and errors × 10^{-6}), by dose series and by age group. The age-group rows show, for Pfizer and Moderna (Janssen) the total number of deaths following the second (first) dose divided by the total number of administered second (first) doses. Estimated 2σ errors in parentheses: two times the square-root of the number of deaths divided by the number of doses.

→ We produce graphs of toxicity (number of AEs / number of doses) by vaccination date or by AE date (not shown), using the independent-database administered dose data, which demonstrate strong correlations of toxicity with median age of those injected on the vaccination or AE dates, and which show a gradation of manufacturer-specific age-accounted toxicity (and see Table 1):

Janssen > Moderna > Pfizer,

approximately in the ratio (deaths per dose)

Janssen : Moderna : Pfizer = 4 : 1.3 : 1

→ We find that the number of deaths per administered dose (e.g., < 60 days since injection) increases exponentially with age, with doubling time ~9-10 years, which is approximately the known doubling time (in lived years) of the mortality rate for adults in the general population of the USA. We interpret this to mean that the same age-dependent repair/immune efficiency is in play defending against the assault of the injection as is active protecting against the usual array of environmental and internal assaults that cause death in adults (see discussion below about batches, and Figure S6).

→ We find that the VAERS deaths by 5-year age groups (per general-population of each USA age group) vary exponentially, again, with a doubling time approximately equal to the known doubling time for risk of death per time (per year) for adults in the general population of the USA. This supports our hypothesis that survival from the assault of the vaccine is determined by the same age-dependent limiting kinetics of the protective repair/immune mechanisms that ensure survival of adults subjected to the current array of dominant life-expectancy-limiting challenges in the USA.

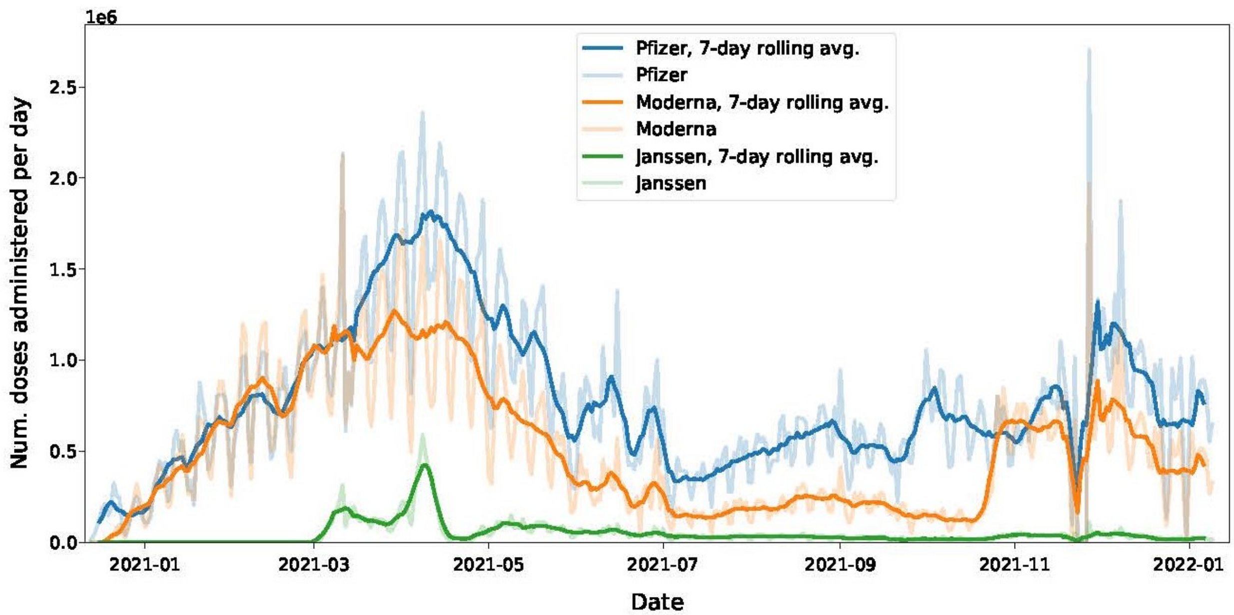
→ We find no evidence that supports the hypothesis of “toxic batches” (batch-to-batch heterogeneity in lethality). The vaccine itself, as designed, is toxic.

→ In looking for “toxic batches”, we instead found natural distributions of age-dependent vulnerability to assault, as follows. Graphs of number of VAERS deaths by batch versus

median age of those who died (per batch) have an upper threshold given by the usual exponential (doubling time ~ 9-10 years), and a breadth of distribution of values that also increases exponentially with age, with approximately the same doubling time (Figure S6). We postulate that this behaviour arises from the natural age-dependent spread of vulnerably to assault, not from batch heterogeneity. Indeed, essentially the same behaviour (exponential increase in spread of sub-sample mortality with age, and similar doubling time) is displayed if we make such plots on the basis of the state jurisdictions or on the basis of vaccination date, rather than on the basis of the batch number (not shown).

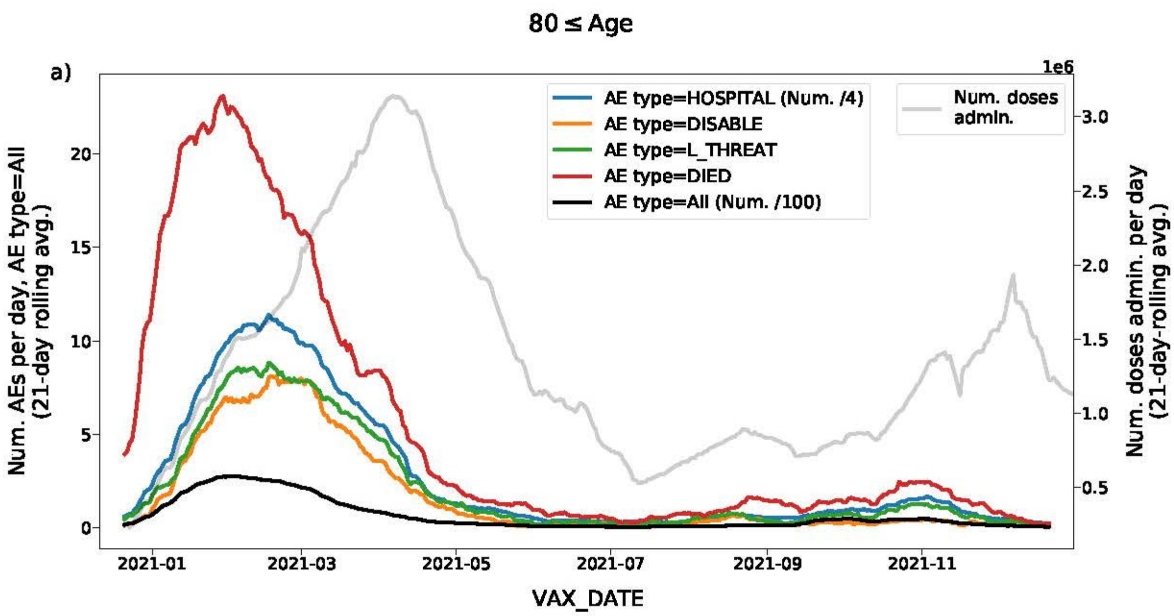
Supporting figures are as follows.

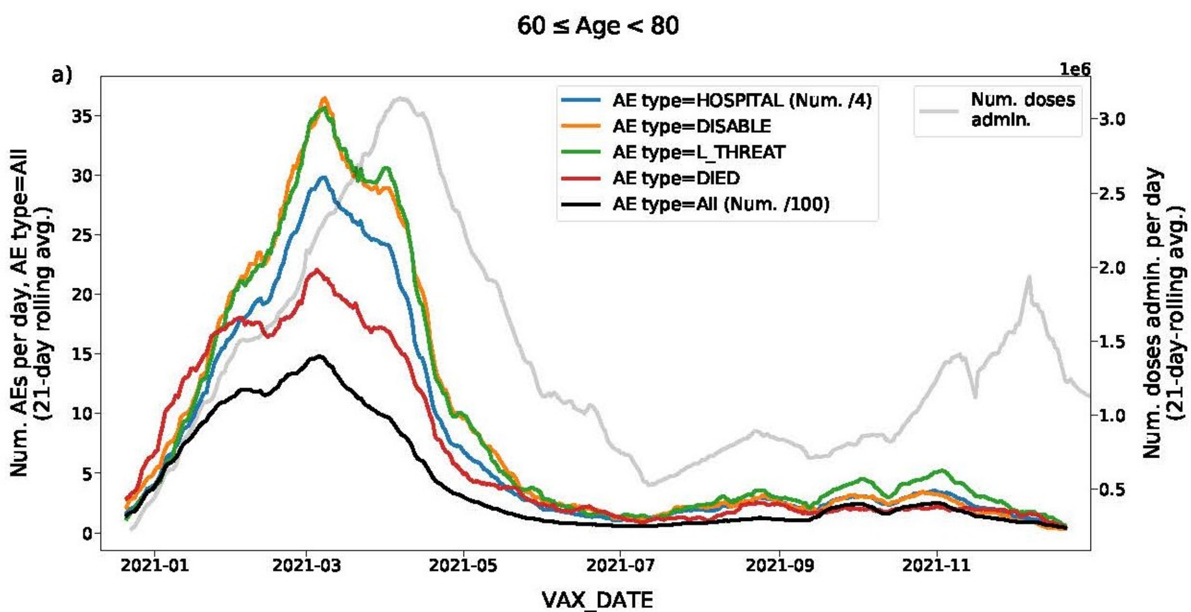
Figure S1. Daily number of doses administered of the Pfizer (blue), Moderna (orange), and Janssen (green) products throughout 2021. Data is from Centers for Disease Control and Prevention (2022). Administered doses show a strong weekly cycle, with fewer doses administered on Sundays. The large dip occurring in December 2021 is due to an artifact present in the CDC data. Details will be given elsewhere. Note: The doses in a primary series, and boosters are also resolved in the data (not shown).

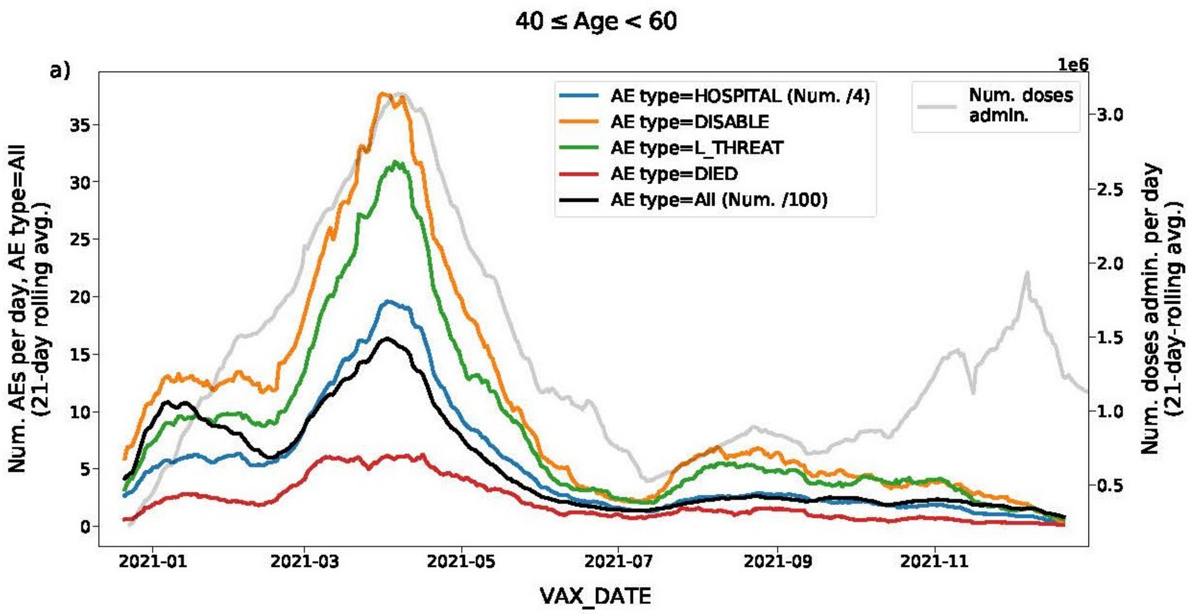


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Figure S2. Number of adverse effects (AEs) of different types (hospitalization, disabled, life-threatening, death, all-AEs, as indicated) per day versus date of vaccination, for different age groups (80+, 60-79, 40-59, 0-39 years, as indicated). Grey curve shows number of doses administered per vaccination date (right y-axes).






40 ≤ Age < 60
a)
AE type=HOSPITAL (Num. /4)
AE type=DISABLE
AE type=L_THREAT
AE type=DIED
AE type=All (Num. /100)
Num. doses admin.
Num. AEs per day, AE type=All (21-day rolling avg.)
Num. doses admin. per day (21-day-rolling avg.)
1e6
VAX_DATE
2021-01
2021-03
2021-05
2021-07
2021-09
2021-11

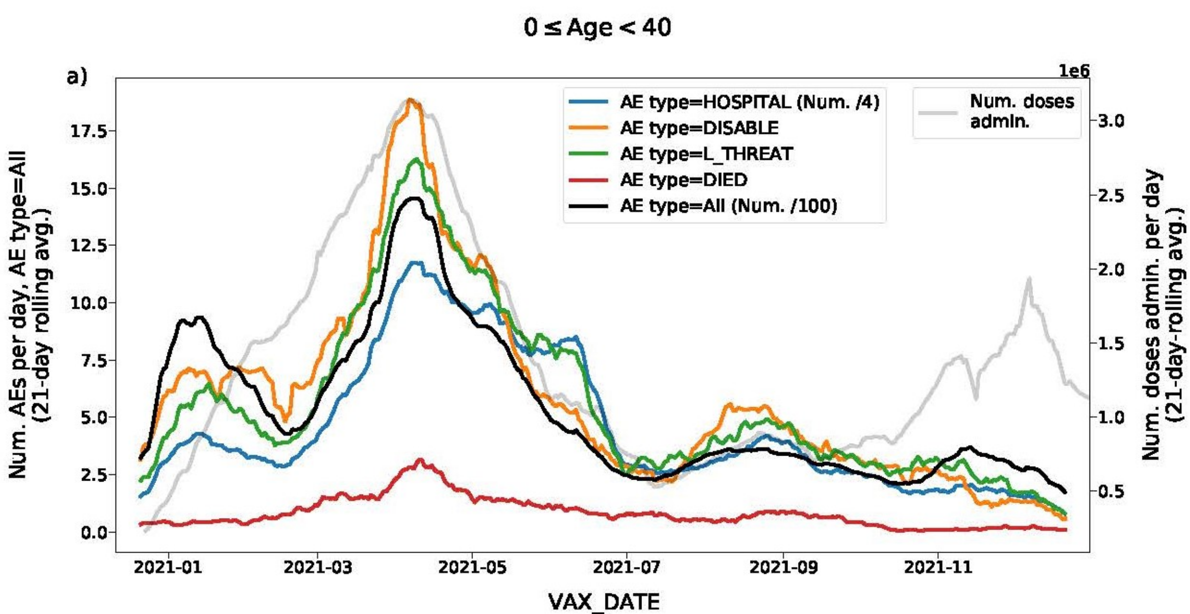

0 ≤ Age < 40
a)
AE type=HOSPITAL (Num. /4)
AE type=DISABLE
AE type=L_THREAT
AE type=DIED
AE type=All (Num. /100)
Num. doses admin.
Num. AEs per day, AE type=All (21-day rolling avg.)
Num. doses admin. per day (21-day-rolling avg.)
1e6
VAX_DATE
2021-01
2021-03
2021-05
2021-07
2021-09
2021-11

Figure S3. Histograms showing the share of VAERS deaths occurring x days after vaccination. (a) shows the full distribution, and its inset shows the same data but zoomed-in on the y-axis. (b) shows the same data but zoomed-in on the x-axis.

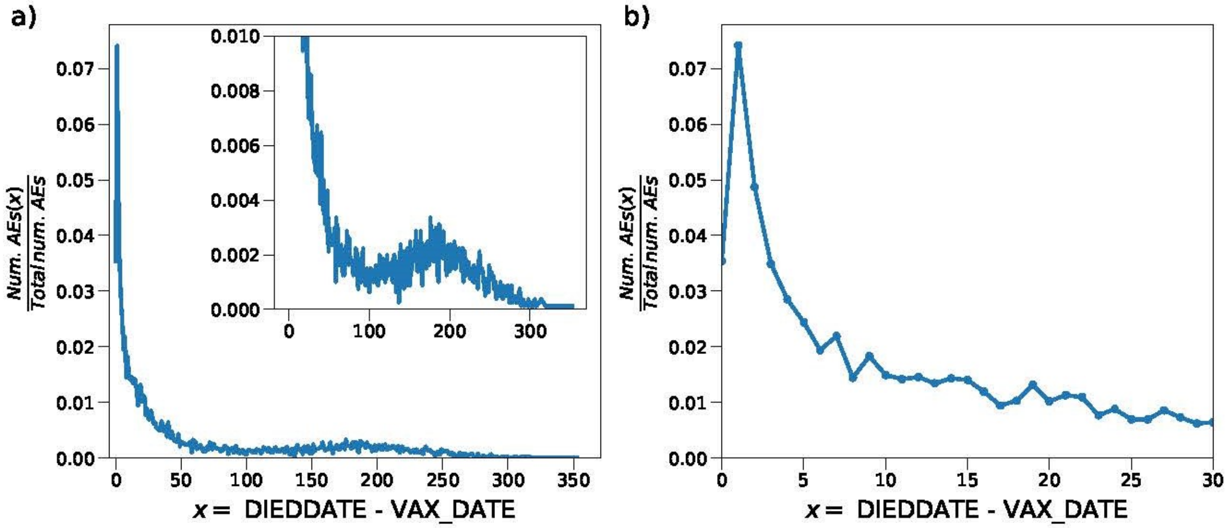


Figure S4. Histograms showing the share of VAERS deaths occurring x days after vaccination, for each manufacturer separately. y-axes are linear on the top row and logarithmic on the bottom row. In the plots in the left column (a and c), deaths at all x values are included in the calculation (but the plots are truncated for better visualization), whereas in the right column (b and d), only deaths for which x < 60 were used. The y-axis in (a) was also truncated for better visualization. Note: The exponential fit (d) gives a half-life equal to 14 days, as indicated.

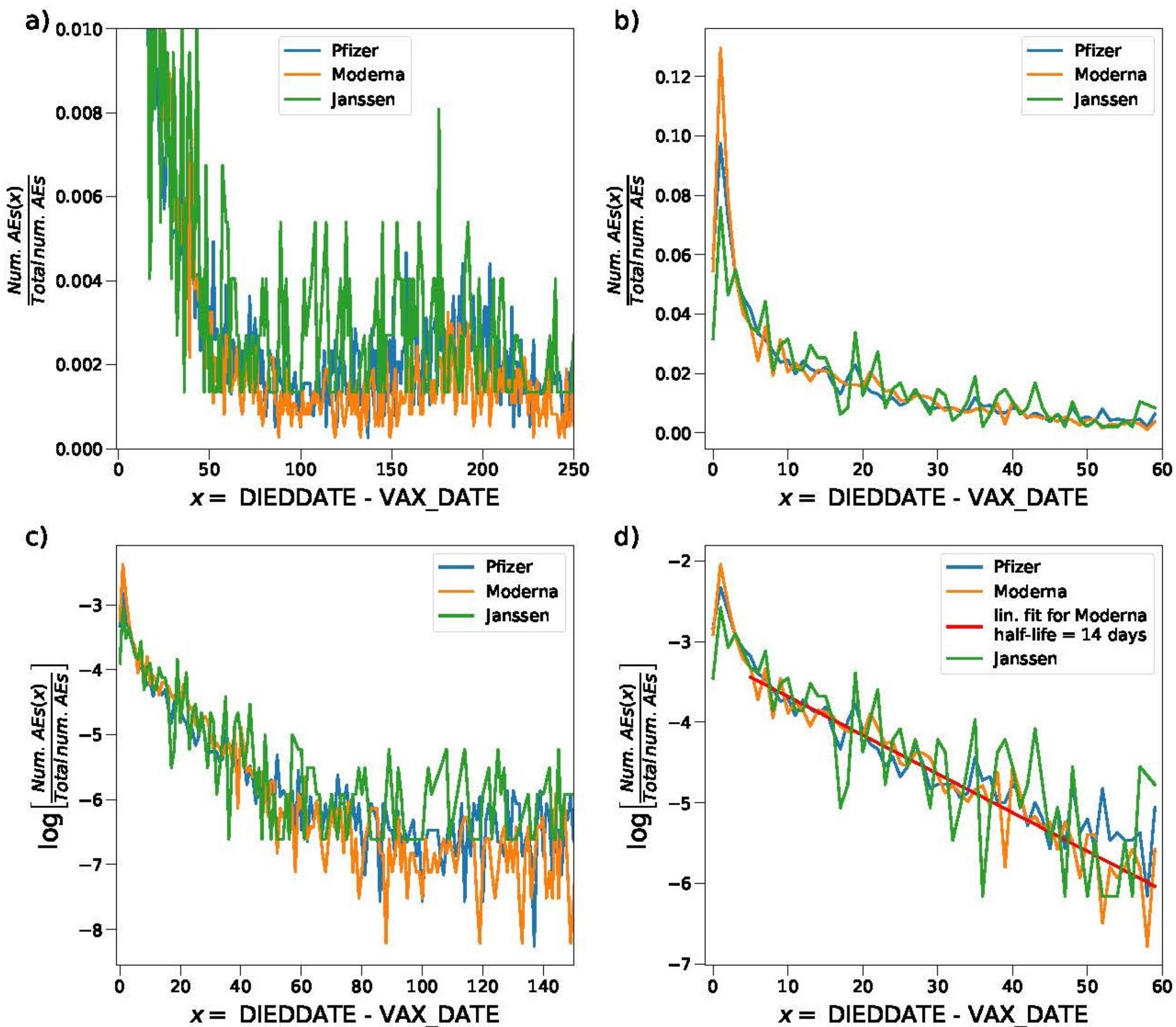


Figure S5. Histograms showing the share of VAERS deaths occurring x days after vaccination, for each manufacturer separately: Pfizer (P) (top row), Moderna (M) (middle row), Janssen (J) (bottom row). The left-most column is for the first dose in a primary series; the second column is for the second dose; and the right-most column is for a third dose. Data for x < 60 days is used. The mean time to death and the total deaths in the graph are as indicated. The exponential fits (red lines) have the following half-life value estimates: 16 days (P1), 25 days (P2), 30 days (P3); 13 days (M1), 21 days (M2), 14 days (M3); 18 days (J1).

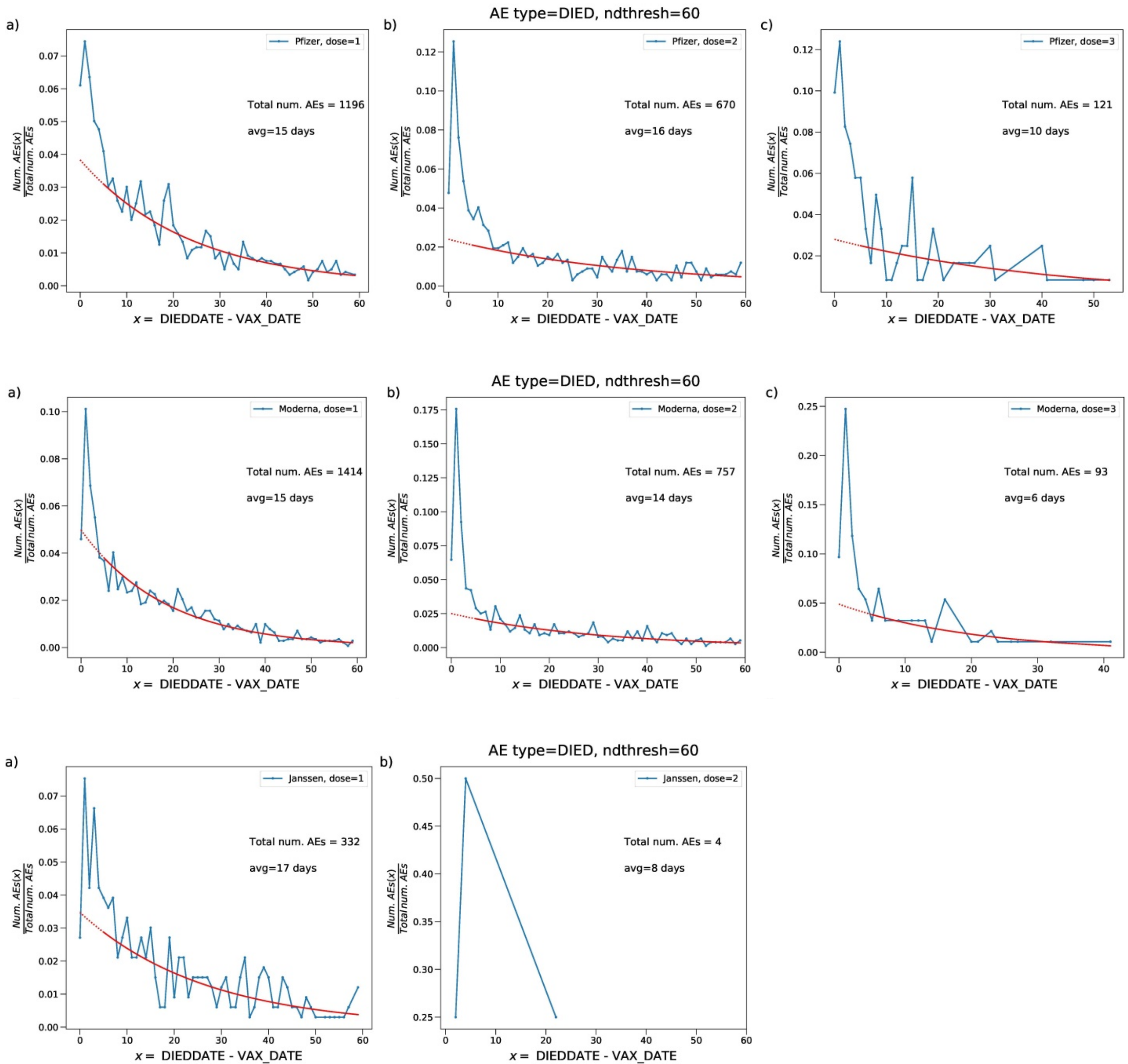
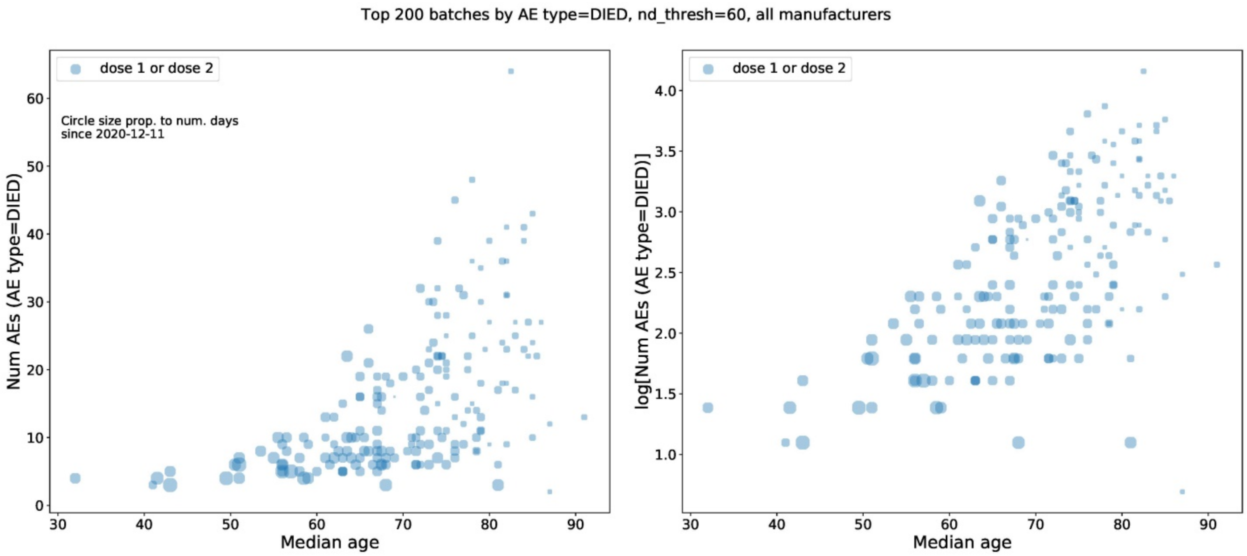


Figure S6. Number of VAERS deaths by batch for the 200 top batches versus median age of those who died (per batch): Linear Y-scale (left), log Y-scale (right). Symbol size is scaled to time (in days) since 11 December 2020.



Nature of the COVID-era public health disaster in the USA, from all-cause mortality and socio-geo-economic and climatic data

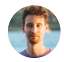
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3 authors, including:



Marine Baudin



Jérémie Mercier

Santé Liberté

Nature of the COVID-era public health disaster in the USA, from all-cause mortality and socio-geo-economic and climatic data

Denis G. Rancourt[1,*], Marine Baudin[2], Jérémie Mercier[2]

[1] Ontario Civil Liberties Association (ocla.ca) ; [2] Mercier Production (jeremie-mercier.com) ;
* denis.rancourt@alumni.utoronto.ca

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25 October 2021

Abstract

We investigate why the USA, unlike Canada and Western European countries, has a sustained exceedingly large mortality in the "COVID-era" occurring from March 2020 to present (October 2021). All-cause mortality by time is the most reliable data for detecting true catastrophic events causing death, and for gauging the population-level impact of any surge in deaths from any cause. The behaviour of the USA all-cause mortality by time (week, year), by age group, by sex, and by state is contrary to pandemic behaviour caused by a new respiratory disease virus for which there is no prior natural immunity in the population. Its seasonal structure (summer maxima), age-group distribution (young residents), and large state-wise heterogeneity are unprecedented and are opposite to viral respiratory disease behaviour, pandemic or not. We conclude that a pandemic did not occur. We infer that persistent chronic psychological stress induced by the long-lasting government-imposed societal and economic transformations during the COVID-era converted the existing societal (poverty), public-health (obesity) and hot-climate risk factors into deadly agents, largely acting together, with devastating population-level consequences against large pools of vulnerable and disadvantaged residents of the USA, far above preexisting pre-COVID-era mortality in those pools. We also find a large COVID-era USA pneumonia epidemic that is not mentioned in the media or significantly in the scientific literature, which was not adequately addressed. Many COVID-19-assigned deaths may be misdiagnosed bacterial pneumonia deaths. The massive vaccination campaign (380 M administered doses, 178 M fully vaccinated individuals, mainly January-August 2021 and March-August 2021, respectively) had no detectable mitigating effect, and may have contributed to making the younger population more vulnerable (35-64 years, summer-2021 mortality).

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Summary

We studied all-cause mortality (ACM) by time (week, year) 2013-2021 for the USA, resolved by state, or by age group, in relation to several socio-geo-economic and climatic variables (poverty, obesity, climatic temperature, population density, geographical region, and summer heatwaves).

We calculate "excess" mortality, by calendar-year or (summer to summer) cycle-year or selected ranges of weeks, as the week-by-week ACM above a summer baseline (SB) ACM, which has a monotonic and linear variation on the decadal timescale, 2013-2019, extrapolated into 2021.

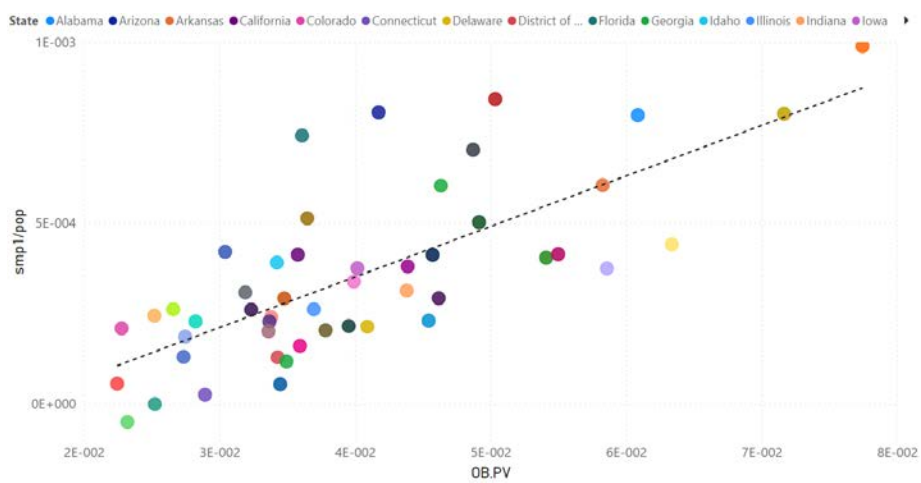
Unlike Canada and Western European countries, the USA has a dramatic anomalous increase in both ACM by year and "excess" ACM by year in 2020 and 2021, which started immediately following the World Health Organization (WHO) 11 March 2020 declaration of a pandemic. Nothing of this magnitude occurs in other nations. The USA's yearly mortality in 2020-2021 is equal to (2020) and greater than (2021) the mortality by year occurring in its domestic population just after the Second World War.

Regarding geo-temporal variations in ACM by week (ACM/w) and in excess (above-SB) ACM by week (ACM-SB/w), we find that there are two distinct periods: the "COVID-era" (March 2020 to present), and the "pre-COVID-era" (prior to March 2020). Normal epidemiological variations occur in the pre-COVID-era, as has been observed for more than a century, in all mid-latitude Northern hemisphere jurisdictions having reliable data; whereas there is unprecedented state-wise jurisdictional and regional geographical heterogeneity in ACM by time in the COVID-era, which is contrary to theoretical pandemic behaviour caused by a new virus for which there is no prior natural immunity in the population.

COVID-era time-integrated seasonal and yearly features of ACM-SB/w significantly correlate with poverty (PV), obesity (OB), and climatic temperature (Tav), by state; and

differ by age group. The correlations account for the state-to-state heterogeneity, with notable outliers in one feature (March-June 2020) of the ACM-SB/w; and such correlations do not occur in pre-COVID-era cycle-year excess mortality. The co-associations of excess deaths with PV, OB and Tav occur only in the COVID-era. We show that normal (pre-COVID) excess (winter season) deaths — largely attributed to viral respiratory diseases occurring in the elderly — occur irrespective of PV, OB and climate, and that there is solely a correlation to age structure of the population in the state.

An example of a co-correlation is the relation between the summer-2020 excess mortality normalized by population (smp1/pop) and the product of OB and PV (OB.PV), state-by-state (see article for details):



A similar large excess of deaths occurred in the summer 2021, which is also strongly co-correlated with poverty, obesity and regional climate. In addition, we showed that these 2020 and 2021 summer mortalities and massive fall-winter-2020-2021 mortality, unlike with viral respiratory disease deaths, occur in younger people, over broad age categories.

In the correlations that we identified, the 2020 and 2021 summer excess (above-SB) mortalities extend to zero values for sufficiently small values of poverty, obesity or

summer temperatures, or their combinations, such as the product of poverty and obesity.

We also found, for example, that the onset of the COVID-era is associated with an increase in deaths of 15-34 year olds to a new plateau in ACM/w (approximately 400 more deaths per week), which does not return to normal over the period studied.

The behaviour of all-cause mortality in the COVID-era is irreconcilable with a pandemic caused by a new virus for which there is no prior natural immunity in the population.

On the contrary, we concluded that the COVID-era deaths are of two types:

- A large narrow peak (in ACM/w) occurring immediately after the WHO declaration of a pandemic apparently caused by the aggressive novel government and medical responses that were applied in certain specific state jurisdictions, against sick elderly populations (34 states do not significantly exhibit this feature).
- Summer-2020, fall-winter-2020-2021, and summer-2021 peaks and excesses (in ACM/w), which co-correlate with poverty, obesity and regional climate, presumably caused by chronic psychological stress induced by the government and medical responses, which massively disrupted lives and society, and affected broad age groups, as young as 15 year olds.

Therefore, a pandemic did not occur; but an unprecedented systemic aggression against large pools of vulnerable and disadvantaged residents of the USA did occur. We interpret that the persistent chronic psychological stress induced by the societal and economic transformation of the COVID-era converted the existing societal (poverty), public-health (obesity) and hot-climate risk factors into deadly agents, largely acting together, with devastating population-level consequences, far beyond the deaths that would have occurred from the pre-COVID-era background of preexisting risk factors.

List of figures

Table of abbreviations and definitions

Abbreviation	Name	Units	Description	Notes
85+	85+	People	Population estimate of people of 85 years old an over as of July 1st of the year	
85+/pop	85+ by population	%	Proportion of the people of 85 years old and older in the population	
ACM	All-cause mortality	Deaths	Total deaths from all causes (occurring in a defined period and for a defined place)	
ACM/w	All-cause mortality by week	Deaths/w	Total deaths from all causes occurring per week	
ACM/w/pop	ACM/w by population	Deaths/w/pop	Total deaths from all causes occurring per week normalized by population	
ACM/y	All-cause mortality by year	Deaths/y	Total deaths from all causes occurring per year	
ACM/y/pop	ACM/y by population	Deaths/y/pop	Total deaths from all causes occurring per year normalized by population	
ACM-SB	All-cause minus summer baseline mortality	Deaths	Difference between total deaths from all causes and deaths from all causes of the summer baseline	1
ACM-SB/w	ACM-SB by week	Deaths/w	Difference between total deaths from all causes and deaths from all causes of the summer baseline per week	
ACM-SB/w/pop	ACM-SB/w by population	Deaths/w/pop	Difference between total deaths from all causes and deaths from all causes of the summer baseline per week normalized by population ("Proportion of excess mortality per week")	
av	Average		Arithmetic mean of all the values of a data set	
(av-med)/av	Average minus median divided by average		Ratio between the difference between the average and the median and the average of the values of a data set	
av-sd	Average minus standard deviation		Difference between the average and the standard deviation of the values in a data set	

COVID-19	Coronavirus disease 2019	N/A	"Coronavirus disease 2019 is a contagious disease caused by severe acute respiratory syndrome coronavirus 2"	
cvp1	COVID-peak 1	Deaths	Integrated deaths of ACM-SB between week 11 of 2020 (week of March 9, 2020) and week 25 of 2020 (week of June 15, 2020), inclusively	2
cvp1/pop	COVID-peak 1 by population	Deaths/pop	COVID-peak 1 normalized by population	
cvp2	COVID-peak 2	Deaths	Integrated deaths of ACM-SB between week 40 of 2020 (week of September 28, 2020) and week 11 of 2021 (week of March 15, 2021), inclusively	3
cvp2/pop	COVID-peak 2 by population	Deaths/pop	COVID-peak 2 normalized by population	
med	Median		The 50th percentile of values in a data set	
neg-cor	Negative correlation			
OB	Obesity	%	Prevalence of self-reported obesity by state and territory (BRFSS (Behavioral Risk Factor Surveillance System), 2020)	
OB.PV	Obesity times poverty		Product of obesity and poverty	
pSB	Pneumonia summer baseline mortality	Deaths	Pneumonia assigned-deaths baseline trend	
Pneumonia-pSB	Pneumonia minus pneumonia summer baseline mortality	Deaths	Difference between total pneumonia-assigned deaths and summer baseline pneumonia-assigned deaths	
PIC	Pneumonia, Influenza and/or COVID-19 mortality	Deaths	Deaths from the following causes: pneumonia and/or influenza and/or COVID-19	
PIC-pSB	PIC minus pneumonia summer baseline mortality	Deaths	Difference between PIC-assigned deaths and summer baseline pneumonia-assigned deaths	
ACM-SB - PIC-pSB	ACM-SB minus PIC-pSB	Deaths	Difference between ACM-SB ("excess") and PIC-pSB ("PIC above pneumonia-baseline") deaths	

pop	Population	People	Resident population estimate for the states of the USA as of July 1st of the year	
popD	Population density	People/mile²	Number of inhabitants per unit surface area (average population per square mile)	
pos-cor	Positive correlation			
PV	Poverty	%	Estimated percent of people of all ages in poverty	
SB	Summer baseline	Deaths	Linear baseline of mortality independent of winter mortality estimated from the summer trough weeks 26 to 37, inclusively, of summers 2013 to 2019, inclusively	
sd	Standard deviation		Measure of the amount of variation or dispersion of values in a data set	
sd/av	Standard deviation divided by average		Ratio between the standard deviation and the average of the values of a data set	
smp1	Summer-peak 1	Deaths	Integrated deaths of ACM-SB between week 26 of 2020 (week of June 22, 2020) and week 39 of 2020 (week of September 21, 2020), inclusively	4
smp1/pop	Summer-peak 1 by population	Deaths/pop	Summer-peak 1 divided by population	
smp2	Summer-peak 2	Deaths	Integrated deaths of ACM-SB between week 26 of 2021 (week of June 28, 2021) and week 37 of 2021 (week of September 13, 2021), inclusively	5
smp2/pop	Summer-peak 2 by population	Deaths/pop	Summer-peak 2 divided by population	
Tav	Average temperature	° F	Average daily average temperature, where an average daily temperature is the average between the max and min daily temperatures	
Tav 2020	Average temperature in 2020	° F	Average daily average temperature over the calendar-year 2020	
Tmax	Maximum temperature	° F	Average daily maximum temperature	
Tmax Jul-Aug 2020	Maximum temperature in July and August 2020	° F	Average daily maximum temperature over July and August 2020	

USA	United States of America	N/A	Here USA means continental USA, which are 49 states, including the District of Columbia and excluding Alaska and Hawaii	
WB	Winter burden	Deaths/y	Integrated deaths of ACM-SB between the week 31 of a year N and the week 30 of a year N+1, inclusively (which is the definition of a cycle-year)	6
WB/pop	Winter burden by population	Deaths/y/pop	Winter burden normalized by population	7

1 Also called "all-cause above-SB" or "excess" deaths in the text
2 Also called "March-June 2020 peak" or "covid peak" or "spring-2020 peak" or "spring-2020 excess mortality" in the text
3 Also called "fall-winter-2020-2021 excess mortality" in the text
4 Also called "summer-2020 excess mortality" in the text
5 Also called "summer-2021 excess mortality" in the text
6 If a year is placed in front, it means it's the WB of this cycle-year
7 If a year is placed in front, it means it's the WB/pop of this cycle-year
N/A stands for not applicable

1. Introduction

A small but growing number of researchers are recognizing that it is essential to examine all-cause mortality (ACM), and excess deaths from all causes compared with projections from historic trends, to make sense of the events surrounding COVID-19 (Jacobson and Jokela, 2021) (Kontopantelis et al., 2021) (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021) (Woolf et al., 2021).

In our prior analyses of ACM by time (by day, week, month, year) for many countries (and by province, state, region or county), we showed that the data in the COVID-era (March 2020 to present) is inconsistent with a viral respiratory disease pandemic, in that the mortality is highly heterogeneous between jurisdictions, with no anomalies in most places, and hot spots or hot regions with deaths that are synchronous with aggressive local or regional responses, both medical and governmental (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021).

The surges in all-cause deaths are highly localized geographically (by jurisdiction) and in time, which is contrary to pandemic behaviour; but is consistent with the surges being caused by the known government and medical responses (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021).

In particular, Canada shows no evidence of a pandemic, since ACM by year (ACM/y) in the COVID-era is squarely on the linear trend of the previous decade. In addition, the ACM by week (ACM/w) data for Canada shows large province-level heterogeneity of temporal and seasonal changes in ACM, by sex and by age group, that must be ascribed to the impacts of medical and governmental measures (Rancourt et al., 2021).

We have also extensively studied ACM by time (day, month, year) for France, at many jurisdictional levels (regions, departments, communes), in comparison to high-resolution

data for institutional occupancies and drug use (Rancourt et al., 2020) (and unpublished), and examined data for European countries, to various degrees of detail.

We reported on the USA in our prior articles about ACM, concentrating on the spectacular hot-spot anomalies that occurred in March through May 2020 (Rancourt, 2020) (Rancourt et al., 2020). Here, we extend our analysis for the USA, up to presently available data, and include socio-geo-economic and climatic data.

The ACM data for the USA in the COVID-era has shocking features, unlike anything else in the world. The USA is unique in this regard. Above-decadal-trend deaths in the COVID-era are massive. Nothing like this occurs in neighbouring Canada. Nothing like this occurs in Western European countries. Similar surges occur in Eastern European countries, but are not of the same large magnitudes as in the USA.

Our goal was to describe the most that can be rigorously inferred from ACM by time, jurisdiction, age group, and sex, in order to elucidate the nature of the massive excess mortality that occurred in the USA in the COVID-era, and delimit its likely causes, with an eye to known mechanisms of disease vulnerability (psychoneuroimmunology, and stress-immune-survival relationships for humans). Therefore, we examined socio-geo-economic data, including:

- Age structure of the population
- Population density
- Racial considerations
- Obesity
- Poverty (also median household income)
- Climatic temperatures
- Vaccination status (COVID-19 and flu vaccines)
- Antibiotic prescription rates

2. Data and methods

Table 1 describes data used in this work and the sources of the data.

Data	Country	Period	Time scale	Filters	Source
ACM	USA	2013-2021*	Week	State	CDC, 2021a
ACM	USA	2013-2021*	Week	Age group[1]	CDC, 2021a
ACM	USA	2020-2021**	Week	Age group[2], sex	CDC, 2021b
ACM	USA	1900-2020[§]	Year	Age group[3], sex	CDC, 2021a CDC, 2021c CDC, 2021d
ACM	USA	1900-1998	Year	Age group[3], sex	CDC, 2021c
ACM	USA	1968-2016	Year	Age group[4], sex	CDC, 2021d
Obesity	USA	2020	Year	State	CDC, 2021e
P-I-C	USA	2013-2021*	Week	-	CDC, 2021a
Population	USA	1900-2020[§§]	Year	Age group[3], sex	CDC, 2021c CDC, 2021d US Census Bureau, 2021b
Population	USA	1900-1997	Year	Age group[5], sex	CDC, 2021c
Population	USA	1968-2016	Year	Age group[4], sex	CDC, 2021d
Population	USA	2010-2020	Year	State	US Census

					Bureau, 2021a
Population	USA	2010-2020[#]	Year	State, age group[6], sex	US Census Bureau, 2021b
Density	USA	1910-2020[##]	Decade	State	US Census Bureau, 2021c
Poverty	USA	2019	Year	State	US Census Bureau, 2021d
Temperature	USA	1895-2021[***]	Month	State[7]	NOAA, 2021
Vaccines	USA	2020-2021[+]	Day	-	CDC, 2021f
ACM	Canada	2010-2021[++]	Week	-	StatCan, 2021

Table 1. Data retrieved. USA means continental USA, composed of 49 states, including the District of Columbia and excluding Alaska and Hawaii, unless otherwise stated in the text.
* At the date of access, data were available from week-40 of 2013 to week-40 of 2021. Usable data are until week-37 of 2021, due to insufficient data in later weeks, which gives a large artifact (anomalous drop in mortality, see Appendix). For the work on USA at the state level, we could add the missing weeks of 2013 (week-1 of 2013 to week-39 of 2020) thanks to a previously downloaded file (downloaded on June 24, 2020) from the same website (CDC, 2021a), which was including those weeks back then.
** At the date of access, data were available from week-1 of 2020 (week ending on January 4, 2020) to week-40 of 2021 (week ending on October 9, 2021). Usable data are until week-37 of 2021 (week ending on September 18, 2021), due to insufficient data in later weeks, which gives a large artifact (anomalous drop in mortality).
*** At the date of access, data were available until August 2021.
[§] These data are a combination of the data found in CDC 2021a, CDC 2021c and CDC 2021d.
[§§] These data are a combination of the data found in CDC 2021c, CDC 2021d and US Census Bureau 2021b.
[#] In our work, we use the population data of the year 2020 (census estimate).
[##] In our work, we use the population density data of the year 2020.
[+] At the date of access, data were available from December 14, 2020 (week-51 of 2020) to September 27, 2021 (week-39 of 2021).

++ At the date of access, data were available from week-1 of 2010 (week ending on January 9, 2010) to week-30 of 2021 (week ending on July 31, 2021). Usable data are until week-20 of 2021 (week ending on May 22, 2021) due to not consolidated data in later weeks, which gives a large artifact (anomalous drop in mortality).
[1] 3 age groups: <18, 18-64, 65+
[2] 11 age groups: <1, 1-4, 5-14, 15-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85+
[3] 12 age groups: <1, 1-4, 5-14, 15-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85+, unknown
[4] 14 age groups: <1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-34, 35-44, 45-54, 55-64, 65-74, 75-84, 85+, not stated
[5] 19 age groups: <1, 1-4, 5-9, 10-14, 15-19, 20-24, 25-29, 30-34, 35-39, 40-44, 45-49, 50-54, 55-59, 60-64, 65-69, 70-74, 75-79, 80-84, 85+
[6] 86 age groups: by 1 year age group, from 0 to 85+
[7] Temperatures are not available for the District of Columbia.

StatCan (2021) defines a death as "the permanent disappearance of all evidence of life at any time after a live birth has taken place" and excludes stillbirths. StatCan specifies that the ACM for 2020 and 2021 is provisional and subject to change, and that the counts of deaths "have been rounded to a neighbouring multiple of 5 to meet the confidentiality requirements of the Statistics Act".

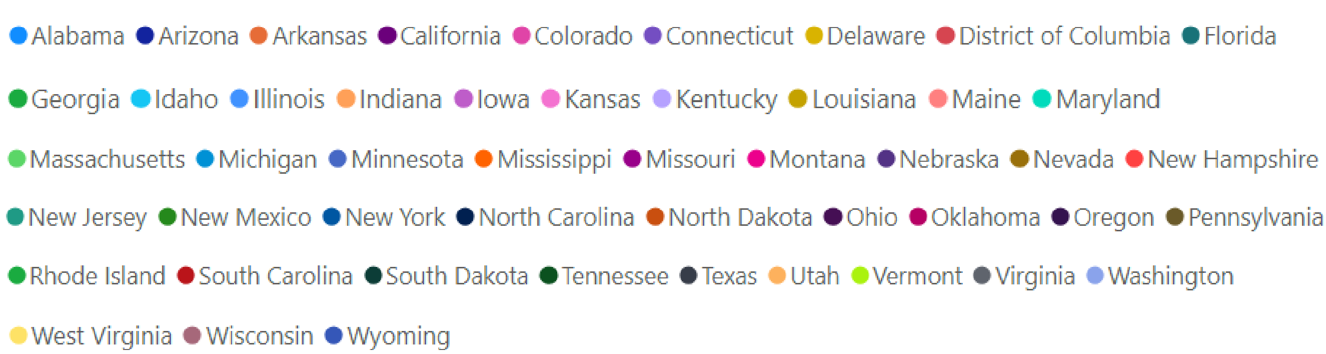
According to CDC (CDC, 2021a):

- "[…] pneumonia, influenza and/or COVID-19 (PIC) deaths are identified based on ICD-10 multiple cause of death codes."
- "NCHS Mortality Surveillance System data are presented by the week the death occurred at the national, state, and HHS Region levels, based on the state of residence of the decedent."
- "Not all deaths are reported within a week of death therefore data for earlier weeks are continually revised and the proportion of deaths due to P&I or PIC may increase or decrease as new and updated death certificate data are received by NCHS."
- "The COVID-19 death counts reported by NCHS and presented here are provisional and will not match counts in other sources, such as media reports or numbers from county health departments. COVID-19 deaths may be classified or defined differently in various reporting and surveillance systems. **Death counts reported by NCHS include deaths that have COVID-19 listed as a cause of**

death and may include laboratory confirmed COVID-19 deaths and clinically confirmed COVID-19 deaths. Provisional death counts reported by NCHS track approximately 1-2 weeks behind other published data sources on the number of COVID-19 deaths in the U.S. These reasons may partly account for differences between NCHS reported death counts and death counts reported in other sources."

- "In previous seasons, the NCHS surveillance data were used to calculate the percent of all deaths occurring each week that had pneumonia and/or influenza (P&I) listed as a cause of death. Because of the ongoing COVID-19 pandemic, COVID-19 coded deaths were added to P&I to create the PIC (pneumonia, influenza, and/or COVID-19) classification. **PIC includes all deaths with pneumonia, influenza, and/or COVID-19 listed on the death certificate**. Because many influenza deaths and many COVID-19 deaths have pneumonia included on the death certificate, P&I no longer measures the impact of influenza in the same way that it has in the past. This is because the proportion of pneumonia deaths associated with influenza is now influenced by COVID-19-related pneumonia. The PIC percentage and the number of influenza and number of COVID-19 deaths will be presented in order to help better understand the impact of these viruses on mortality and the relative contribution of each virus to PIC mortality."

For all the scatter plots presented in this article, the following colour-code is applied for the 49 continental states of the USA (including District of Columbia, excluding Alaska and Hawaii).



The main points of our methodology are as follows.

We work with all-cause mortality (ACM), deaths from all causes, in order to avoid the uncertainty and bias in attributing a cause of death, in this context of COVID-19 in which cause of death is not simple nor obvious. ACM data is available by jurisdiction (state, country, county), by age group, by race, by sex, and by time (day, week, year). We can normalize group-specific ACM totals by the respective populations of the relevant groups, in order to allow comparisons between jurisdictions or different groups, on a per-population basis.

Generally, in jurisdictions that exhibit seasonal winter maximums of mortality, the bottom-values of mortality in the summer troughs follow a straight-line trend on a decadal or shorter timescale. We call this trend-line the “summer baseline” (SB), and we use it to count above-SB deaths, when we wish to thus quantify “excess deaths”.

In other words, we are following our previous methodology in which we argued that mortality by time (day, week, month) is best analyzed using a SB, and winter burden (WB) deaths above the SB, over a (natural) cycle-year from summer to following summer, rather than use assumed underlying sinusoidal seasonal variations of any presumed component(s), since such sinusoidal theoretical curves fail to represent the data or any of its inferred principle components (e.g., Simonsen et al., 1997). Although the summer trough mortality values follow a linear local trend by time (in normal, pre-COVID-era, circumstances), above-SB features have significant randomness in their season to season variations, suggesting that summer baseline mortality is representative of “stable” mortality not influenced by the many different and seasonally variable winter-time life-threatening health challenges (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021).

SB estimation at the state level

The linear summer baseline (SB) is a least-squares fit to the summer troughs for summer-2013 through summer-2019, using the summer trough weeks 27 to 36,

included, for all the states of the continental USA, except for Alabama and Wisconsin for summer-2014 and summer-2015, respectively, and corrected by 1 % (see below). For Alabama, only the weeks [30-32] were used for summer-2014 as drops in data are seen for weeks [27-29] and weeks [33-36] of 2014 (see Appendix). For Wisconsin, only the weeks [27-29] and [33-36] were used for summer-2015 as a drop in data is seen for weeks [30-32] of 2015 (see Appendix). We corrected the SB by 1 % so as to lower the SB and make it match the bottoms of the summer troughs. We also estimated the SB taking different summer periods, from the shortest to the largest: weeks [30-32], weeks [29-33], weeks [28-35] and weeks [27-36], to determine our 1 % correction. We found that the larger the period, the better the estimate of the SB slope, but also the higher the estimate of the SB intercept, as the last weeks towards the previous winter season and the first weeks towards the next winter season are included. We thus decided to estimate the SB with the largest summer period (weeks [26-37]) and lower the intercept by 1 % (no correction leading to a too high intercept and a correction factor of 2 % leading to a too low intercept). The SB is so estimated between the weeks 26 and 37 (inclusively) of each summer of the pre-COVID-era (summers 2013 to 2019), which corresponds to the weeks laying from the beginning of July to the beginning of September.

SB estimation at the national level

- For work involving the states, the SB estimate of the USA is a sum of the SB estimates of each individual state.
- For work not involving the states, the SB is a least-squares fit to the summer troughs for summer-2014 through summer-2019, using the summer trough weeks 27 to 36, included, for the whole USA (including Alaska and Hawaii) with no correction, since none was needed.

In the same way that we thus quantify a winter burden of deaths in a given cycle-year, we can also quantify an excess (above-SB) of deaths over any period of time, such as over a period that captures any prominent features in ACM by time. We defined such periods of interest occurring in the COVID-era: a spring-2020 peak (cvp1),

summer-2020 (smp1), the fall-winter-2020-2021 maximum (cvp2), and summer-2021 (smp2), as specified in the text.

3. Results, analysis and discussion

3.1. All-cause mortality per year, USA, 1900-2020

We start by examining ACM/y (per calendar-year) in the USA, for the years 1900 through 2020. This is shown in Figure 1.

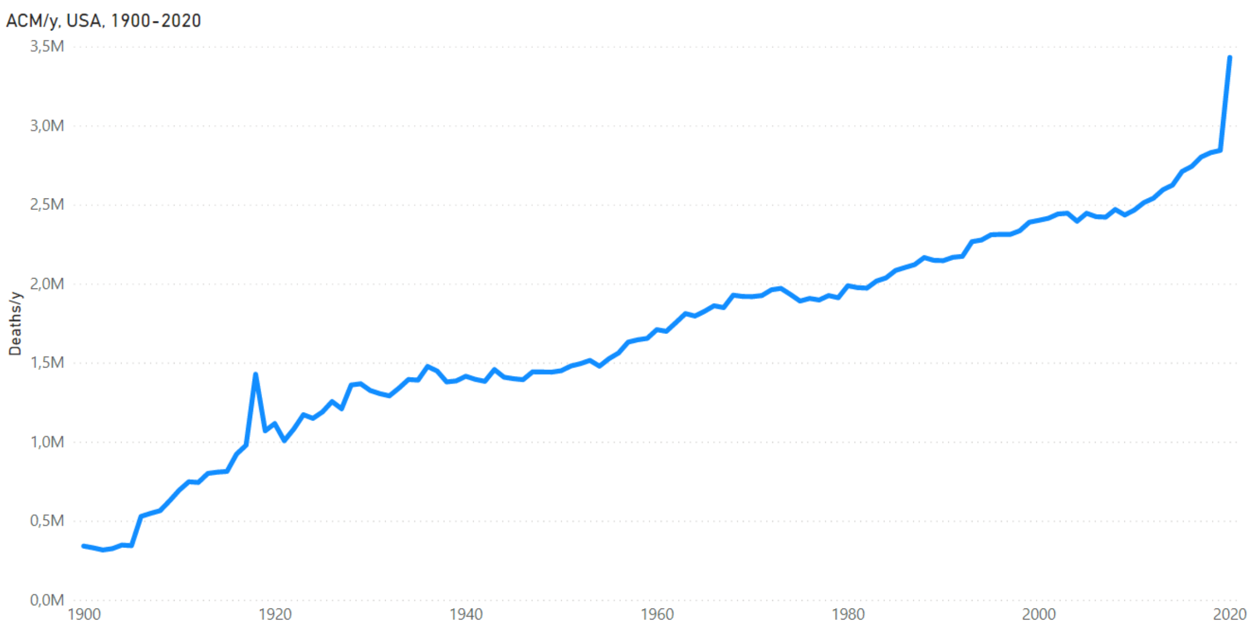


Figure 1. All-cause mortality by calendar-year in the USA from 1900 to 2020. Data were retrieved as described in Table 1.

The ACM/y 1900-2020 has the following main features. First, it has a generally increasing trend over the entire period, with a slope of approximately 16K deaths per year per year (16K/y/y) in the region 1920-2010. The overall increasing trend is due to population growth. One needs to normalize by population to remove this dominant effect (see below). Second, there is a large increase in 1918, which corresponds to the so-

called “1918 Flu Pandemic”. Third, there is a large increase in 2020, which corresponds to the first year of the COVID-era. Fourth, there are notable increases in the late-1920s and mid-1930s, which correspond to the hardships associated with The Great Depression and the accompanying decade-long Dust Bowl droughts of the Midwest. Fifth (by omission), there are no detected increases that would correspond to any of the major 20th-21st century influenza pandemics that are described to have occurred in 1957-58, 1968, and 2009 (Doshi, 2008) (Doshi, 2011).

These main features in ACM/y are clarified and enhanced on examining ACM/y by age group (available for 1900-2016). This is shown for all the ages, excluding <1 year, divided into 10 age groups in Figure 2.

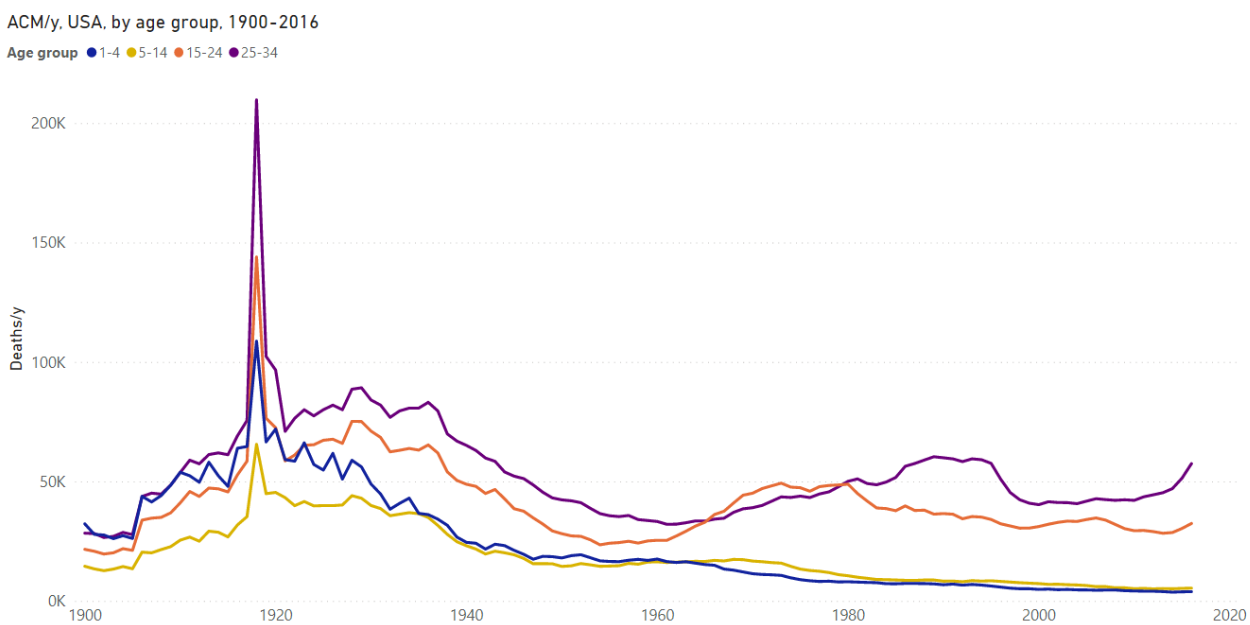


Figure 2a. All-cause mortality by year in the USA for the 1-4, 5-14, 15-24 and 25-34 years age groups, from 1900 to 2016. Data are displayed per calendar-year. Data were retrieved as described in Table 1.

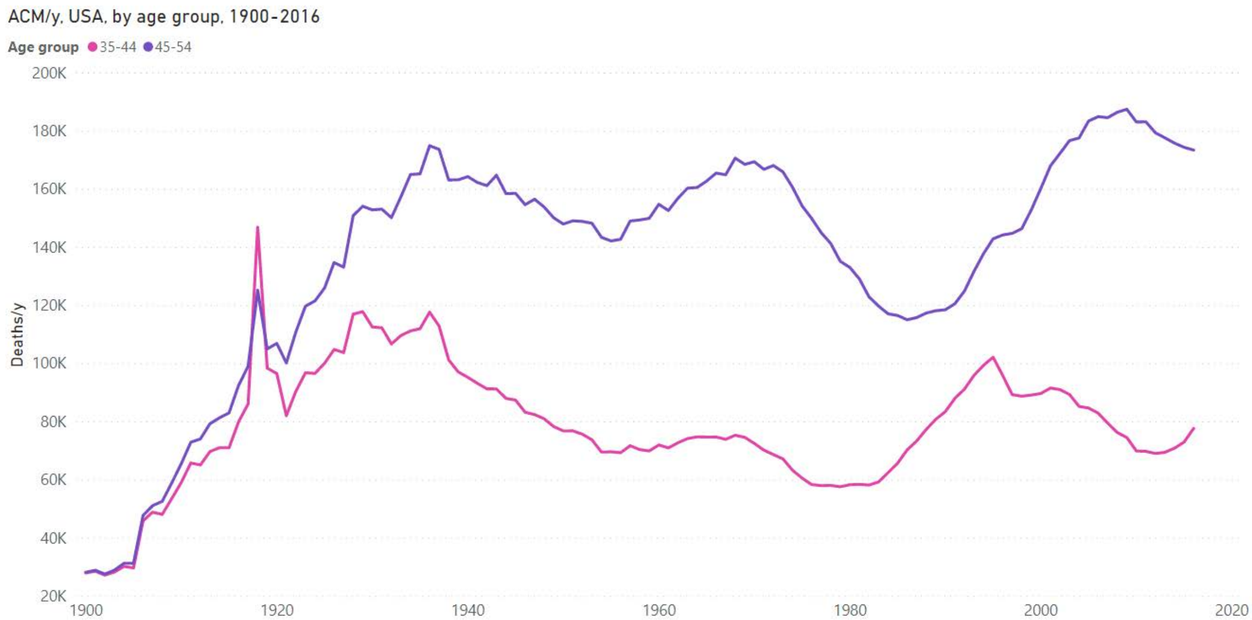


Figure 2b. All-cause mortality by year in the USA for the 35-44 and 45-54 years age groups, from 1900 to 2016. Data are displayed per calendar-year. Data were retrieved as described in Table 1.

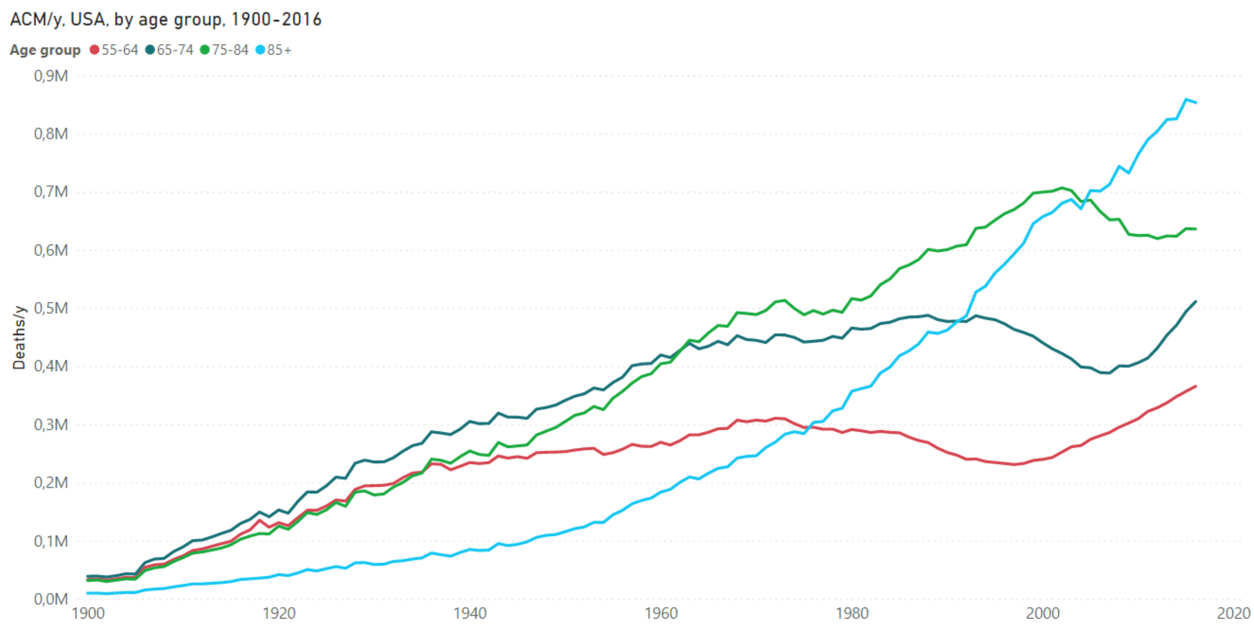


Figure 2c. All-cause mortality by year in the USA for the 55-64, 65-74, 75-84 and 85+ years age groups, from 1900 to 2016. Data are displayed per calendar-year. Data were retrieved as described in Table 1.

The ACM/y 1900-2016 by age-group data allows the following observations to be made.

Regarding 1918, the event was devastating for the age groups 15-24 years and 25-34 years, much less so for the age groups 35-44 years and 45-54 years, and virtually undetected for those 55 years and older, which would be very surprising for influenza. In fact, we know that most of the deaths were associated with massive bacterial lung infections (Morens et al., 2008) (Chien et al., 2009) (Sheng et al., 2011), in an era predating antibiotics, in a period massively perturbed by a world war, and that the event was concomitant with typhoid epidemics in Europe and Russia.

Regarding The Great Depression and the Dust Bowl devastation, the late-1920s and mid-1930s increases in ACM/y are prominent for the 15-24, 25-34, 35-44 and 45-54 years age groups, but are not detected for 55 year olds and older.

Regarding 20th-21st century purported influenza pandemics, there is no trace of increased mortality for 1957-58, 1968, and 2009, in any age group, including the older age groups of 55-64, 65-74, 75-84, and 85+ years. Clearly, these 20th century declared pandemics had negligible impacts on all-cause mortality; not comparable to the large impacts of the events of 1918, late-1920s-mid-1930s, <1945, and 2020, which are associated with major socio-economic upheavals (the First World War, The Great Depression and Dust Bowl, the Second World War, and the medical and government response to the declared COVID-19 pandemic, respectively).

The ACM/y by age group has long-period (decadal) variations with notable broad minima occurring at approximately:
~1975-1980: 35-44 years age group
~1985-1990: 45-54 years age group
~1995-2000: 55-64 years age group
~2005-2010: 65-74 years age group
~2010-2015: 75-84 years age group
These variations are due to the post Second World War baby boom effects on population.

The population of the USA varied from 1900 to 2020 as shown in Figure 3 (and from 1900 to 2016 for the age groups).

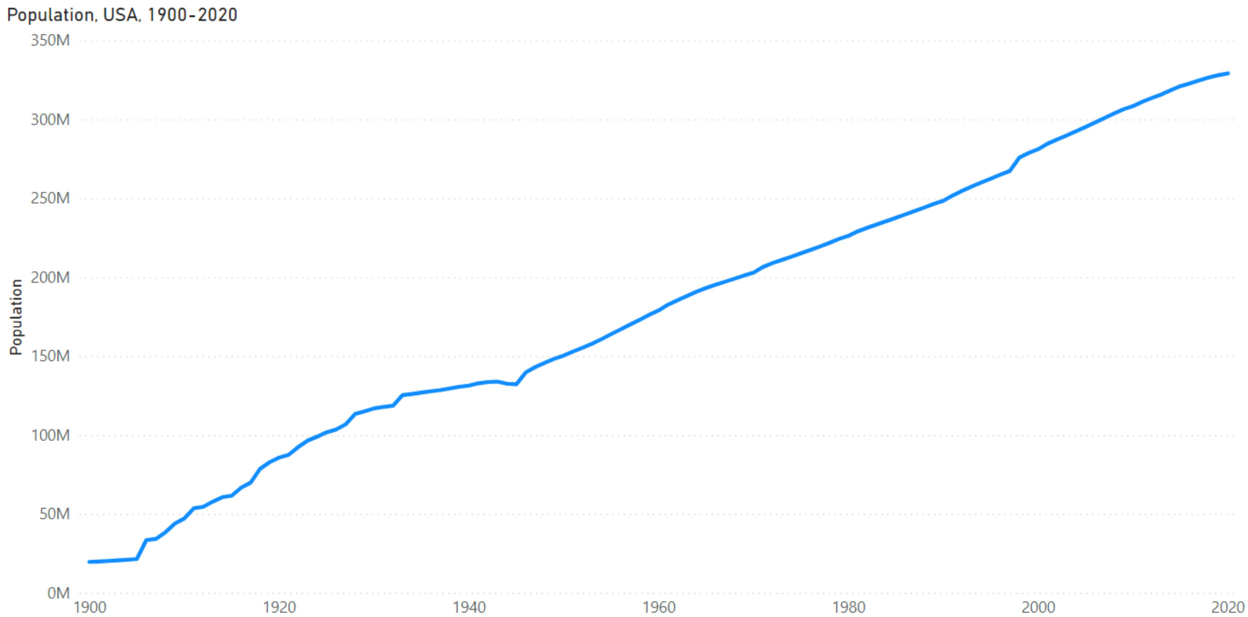


Figure 3a. Population of the USA from 1900 to 2020. Data are displayed per calendar-year. Data were retrieved as described in Table 1.

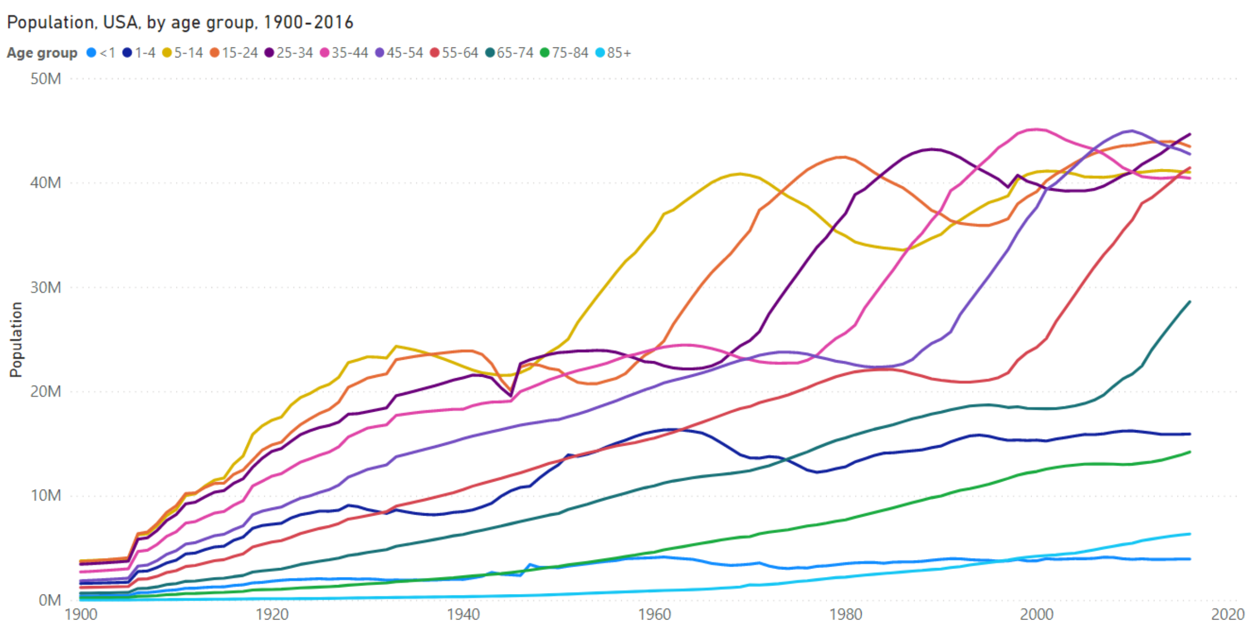


Figure 3b. Population of the USA by age group from 1900 to 2016. Data are displayed per calendar-year. Data were retrieved as described in Table 1.

Here (Figure 3a), we see a large dip in population at 1943-1945, related to the Second World War. The slope to population versus time also changes dramatically at 1943-1945, increasing after the war, in accordance with the known baby boom. The population by age group (Figure 3b) confirms that the dip at 1943-1945 is solely from the 15-24 and 25-34 years age groups, especially 15-24 years. This figure (Figure 3b) also shows the dramatic consequences of the baby boom, showing itself, age group after age group, as the baby boomers age. The monotonic increase in the 85+ years population (Figure 3b) is directly the cause of the monotonic increase in 85+ years deaths (Figure 2c).

Next, we normalize ACM/y (Figure 1) by population (Figure 3a), 1900-2020, to obtain ACM/y/pop shown in Figure 4a.

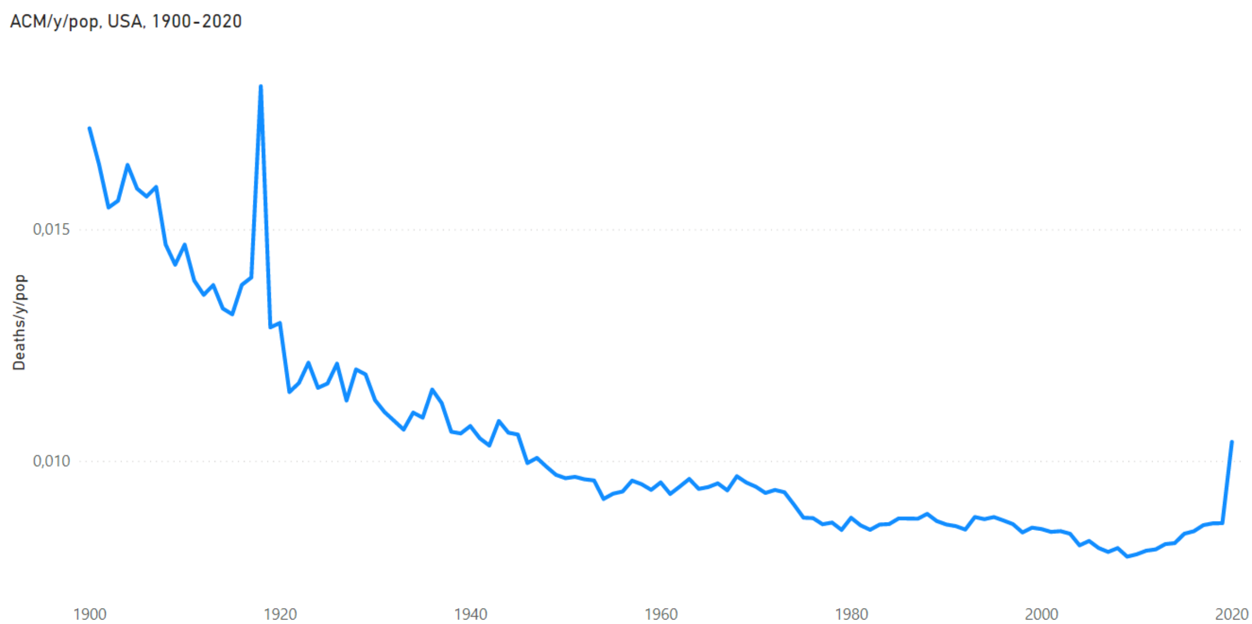


Figure 4a. All-cause mortality by year normalized by population for the USA from 1900 to 2020. Data are displayed per calendar-year. Data were retrieved as described in Table 1.

This allows us to see ACM/y expressed as a fraction of population. We again see the gigantic catastrophe that was the 1918 event (pneumonia/typhoid, wartime upheaval), peaks in the late-1920s and mid-1930s (Great Depression, Dust Bowl), a peak in the Second World War period (young men, 15-24 and 25-34 years age groups, as per

Figure 3b), relatively uneventful mortality after 1945 (no public health catastrophes detected), no sign of the announced pandemics of 1957-58, 1968, and 2009, and the COVID-era increase of 2020 (a subject of this article).

The mortality events of the late 1920s, mid-1930s and <1945, and the >1945 uneventful period, are elucidated further by examining ACM/y/pop resolved by age group and by sex, as per the following.

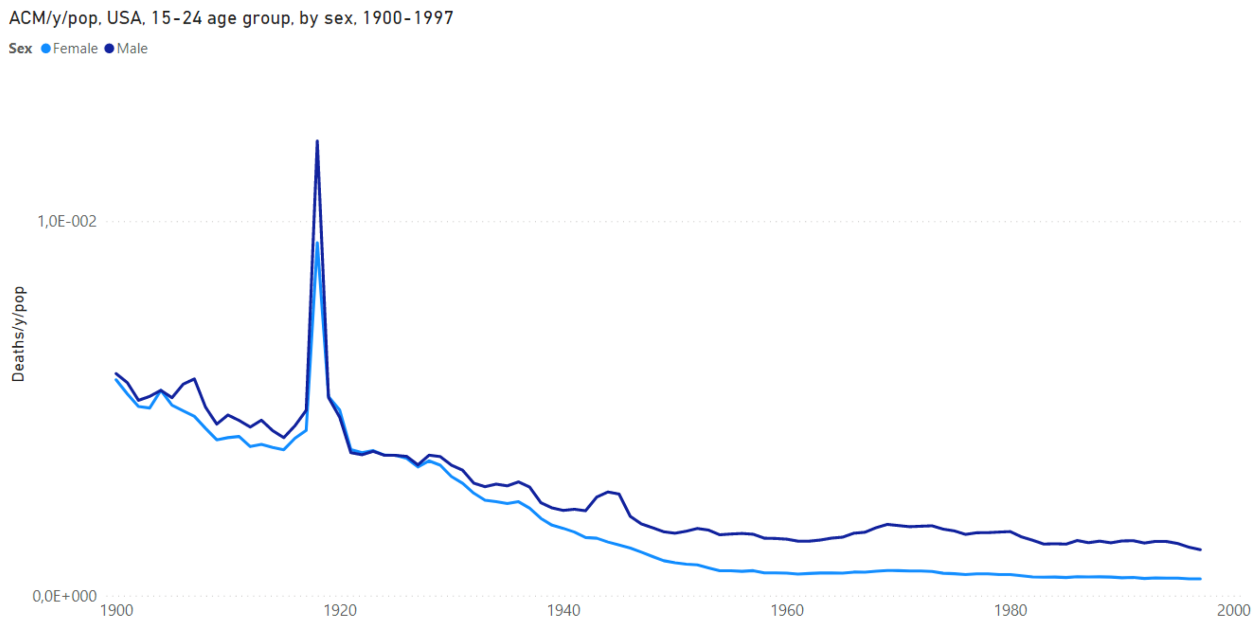


Figure 4b. All-cause mortality by year normalized by population for the USA for the 15-24 years age group, for each of both sexes, from 1900 to 1997. The population of the specific age group and sex is used for each normalization. Data are displayed per calendar-year. Data were retrieved as described in Table 1.

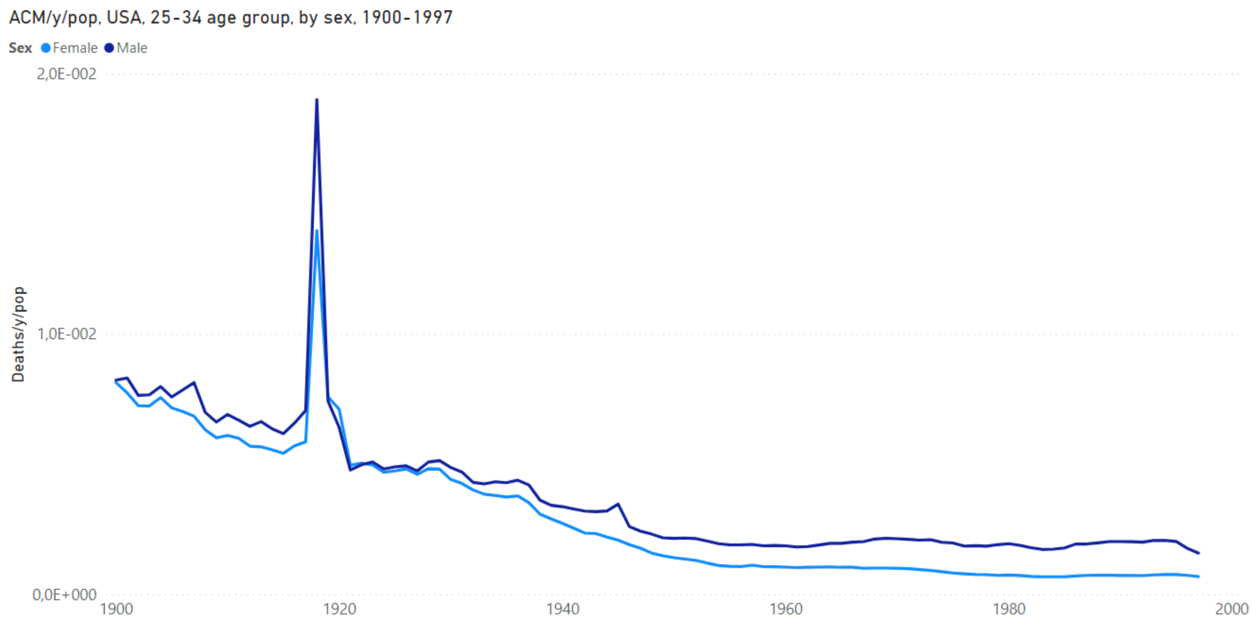


Figure 4c. All-cause mortality by year normalized by population for the USA for the 25-34 years age group, for each of both sexes, from 1900 to 1997. The population of the specific age group and sex is used for each normalization. Data are displayed per calendar-year. Data were retrieved as described in Table 1.

Figures 4b and 4c show that both young men and women were impacted by the hardships of the late-1920s and mid-1930s, but that only young men were impacted to death by the Second World War. Interestingly, 15-24 year old men had relatively high mortality between the mid-1960s and the early-1980s.

The 2020 value of ACM/y/pop brings us back to a mortality equal to the mortality by population that prevailed in 1945 (Figure 4a), which suggests that the socio-economic upheavals from COVID-19 response are comparable to the upheavals from the last major war period, with an albeit much older population presently, and possibly greater class disparity, since The New Deal had already been implemented in 1945, in response to the hardships of the 1930s.

3.2. ACM by week (ACM/w), USA, 2013-2021

The ACM/w for the USA from 2013 to 2021 is shown in Figure 5, with a straight-line trend for the bottoms of the summer troughs for 2013 through 2019 (of the pre-COVID-era). We call this trend-line the "summer baseline" (SB), and we use it to count above-SB deaths ("excess" deaths).

We are following our previous methodology in which we argued that mortality by time (day, week, month) is best analyzed using a SB, and winter burden deaths (WB) above the SB, over a (natural) cycle-year from summer to following summer, rather than use assumed underlying sinusoidal seasonal variations of any presumed component(s), since such sinusoidal theoretical curves fail to represent the data or any of its inferred principle components (e.g., Simonsen et al., 1997). It is a general feature with seasonal mortality data that SB trends are typically linear on the timescale of one decade or so, whereas above-SB features have significant randomness in their season to season variations, suggesting that summer baseline mortality is representative of "stable" mortality not influenced by the many different and seasonally variable winter-time life-threatening health challenges (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021).

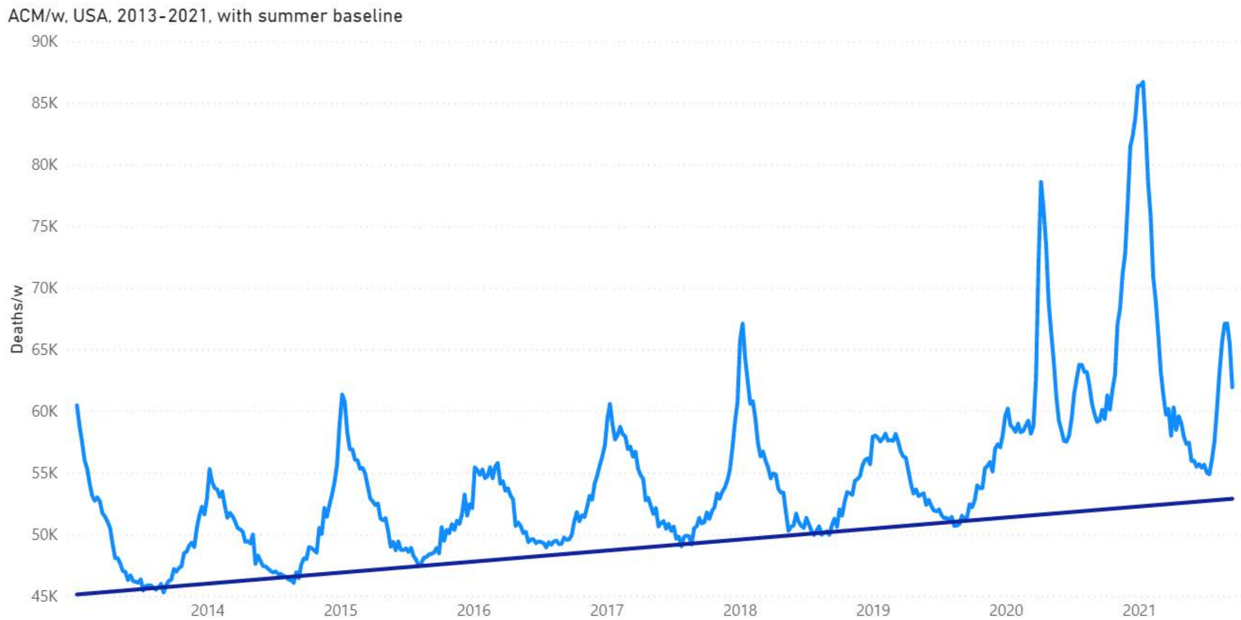


Figure 5. All-cause mortality by week in the USA from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The linear summer baseline (SB) is a least-squares fit to the summer troughs for summer-2013 through summer-2019, using the summer trough weeks 27 to 36, included, except for Alabama and Wisconsin for summer-2014 and summer-2015, respectively, and corrected by 1 % (see section 2). Data were retrieved from CDC (CDC, 2021a), as described in Table 1.

Next, for the sake of visualization, we can remove the SB from the ACM, week by week, to obtain ACM-SB/w. This is shown for the USA from 2013 to 2021, in Figure 6, where we have used different colours for the different cycle-years.

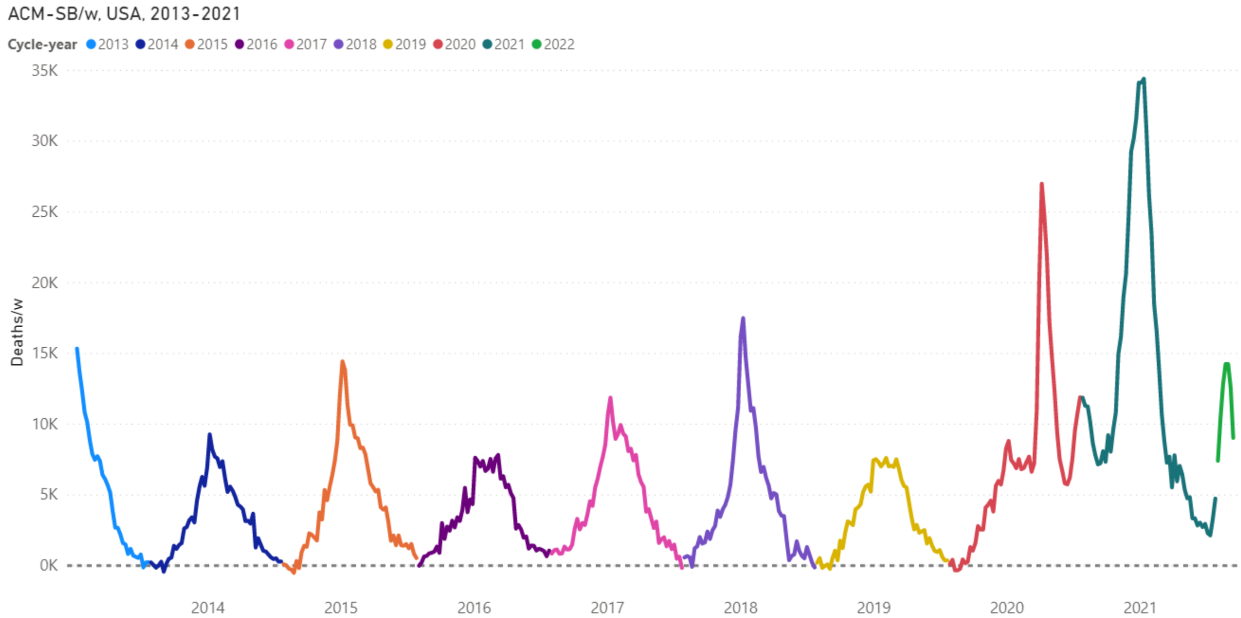


Figure 6. Difference between all-cause mortality and summer baseline mortality for the USA from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The different colours are for the different cycle-years. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). ACM data were retrieved from CDC (CDC, 2021a), as described in Table 1. SB was estimated as described in section 2.

Many striking features occur in ACM/w (or ACM-SB/w) in the COVID-era period for the USA (Figures 5 and 6):

- The WB (total above-SB deaths per cycle-year) is much greater in cycle-years 2020 (summer-2019 to summer-2020) and 2021 (summer-2020 to summer-2021) than in cycle years 2014 through 2019, which is consistent with ACM/y already discussed above (Figures 1 and 4).
- The 2020 cycle-year exhibits a sharp and intense feature spanning weeks 11 through 25 of 2020, starting when the pandemic was declared by the World Health Organization (WHO) on 11 March 2020, lasting three months, and which we have called "the COVID peak" and amply described in our previous articles (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021). In this article, we refer to this feature and its integrated intensity as "cvp1".
- There is "no summer", in terms of lower mortality, in the summer-2020. The ACM/w does not descend down to the SB. In fact, the summer of 2020 exhibits a

broad mid-summer peak in ACM/w, spanning weeks 26 through 39 of 2020 (approximately mid-June to mid-September), which is unprecedented in any ACM by time data that we have examined, for data since 1900 for dozens of countries and hundreds of jurisdictions. In this article, we refer to this feature and its integrated intensity as "smp1".

- The 2021 cycle-year exhibits a massive peak, spanning from week-40 of 2020 through to week-11 of 2021 (approximately late-September 2020 to mid-March 2021). The peak extends to 35K deaths per week above SB. It is anticipated that the ACM/y for 2021 will be larger than for 2020, which in turn brought us back to mortality of the magnitude that was occurring just after the Second World War, on a per population basis (Figure 4a). In this article, we refer to this winter 2020-2021 feature and its integrated intensity as "cvp2".
- Finally, there is a summer-2021 upsurge of mortality (ACM/w) in the last weeks of the usable data set, starting in mid-July 2021. This upsurge in ACM/w is particularly large for Florida, for example. We refer to this feature as "smp2", which is interrupted by the end of the data set (week-37 of 2021 for consolidated data, as described in section 2).

To be clear, the three uninterrupted prominent features in the USA ACM/w for the COVID-era (cvp1, smp1, and cvp2) are shown, according to their operational definitions in Figure 7. For each feature, its quantification is achieved by summation of ACM-SB/w over the weeks spanned by the feature. The late-summer-2021 feature "smp2" is also indicated.

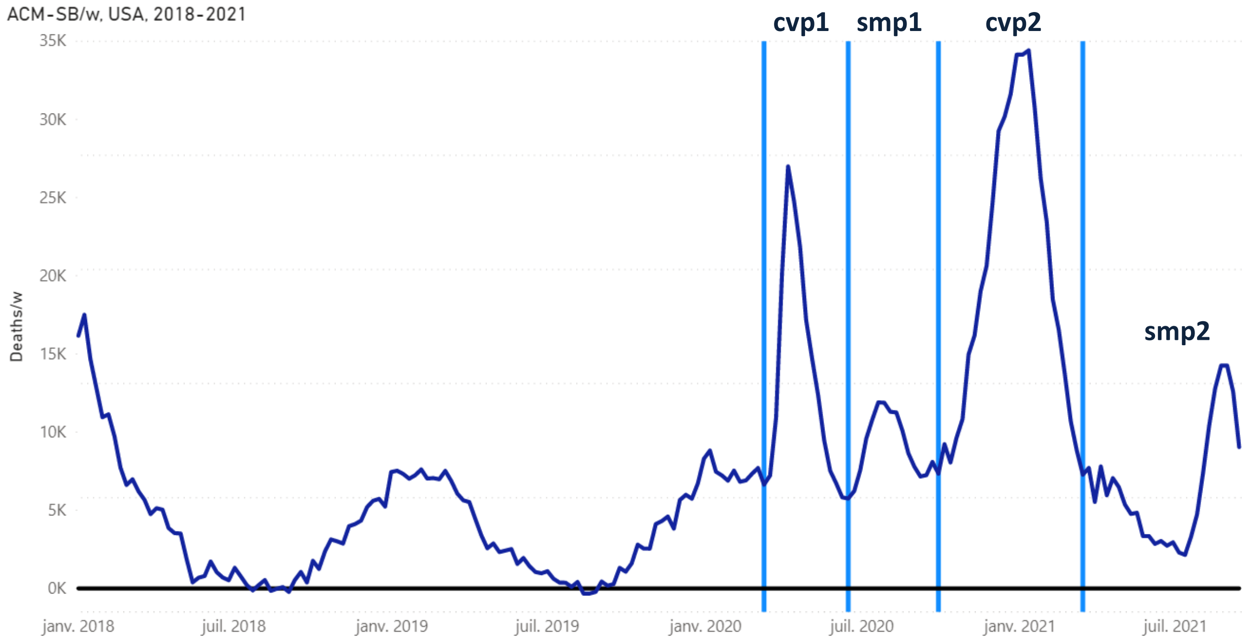


Figure 7. Difference between all-cause mortality and summer baseline mortality for the USA from 2018 to 2021. Data are displayed from week-1 of 2018 to week-37 of 2021. The cvp1, smp1, cvp2 and smp2 features discussed in the text are indicated. The light-blue vertical lines represent the weeks 11, 25, 40 of 2020 and 11 of 2021, emphasizing the delimiting weeks of the cvp1, smp1 and cvp2 features. The constant zero line is in black. ACM data were retrieved from CDC (CDC, 2021a), as described in Table 1. SB was estimated as described in section 2.

Although these features in USA ACM (cvp1, smp1, cvp2, smp2; highlighted in Figure 7) are unprecedented in recent decades and are shocking in themselves; an equally striking aspect is only seen on examining ACM/w (or ACM-SB/w) by state, for individual states. The later examination shows (below) that the said features in the COVID-era, unlike anything previously observed in epidemiology, are often dramatically different, in both relative and absolute magnitudes, and in shape and position, in going from state to state. The next section is devoted to illustrating this remarkable state-to-state variability in COVID-era ACM by time.

3.3. ACM by week (ACM/w), USA, 2013-2021, by state

Graphs of ACM/w, from 2013 to 2021, with colour-differentiated cycle-years, for all the individual states of continental USA (excluding Alaska and Hawaii) are shown in Appendix (attached below).

In these graphs (Appendix), note that the pre-COVID-era seasonal pattern (2013-2019) is essentially identical from state to state (more on this further below), whereas there are large state to state changes in the COVID-era patterns. This concurs with our previous findings that COVID-era behaviour in ACM by time is abnormally heterogeneous on a jurisdictional basis, which is the opposite of past seasonal epidemiological behaviour (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021). Woolf et al. (2021) also report large USA regional differences in all-cause excess mortality by time patterns during the COVID-era.

Some comparative and systematic features in these curves (Appendix) are as follows.

- **L0M / North-Easterly coastal states:** Several of the North-Easterly coastal states exhibit a pattern in cvp1-smp1-cvp2 (an “L0M” pattern) in which cvp1 is very large, smp1 is essentially zero (ACM/w comes down to the SB values) and cvp2 is of medium magnitude: New York, New Jersey, Connecticut, Massachusetts and Rhode Island, and Maryland and District of Columbia to some degree.
- **LSL / North-Central-Easterly non-coastal states:** A group of neighbouring North-Central-Easterly non-coastal states exhibit a pattern in cvp1-smp1-cvp2 (an “LSL” pattern) in which cvp1 is large, smp1 is small (near-zero) and cvp2 is large: Colorado, Delaware, Illinois, Indiana, Michigan, and Pennsylvania, although Michigan has a unique extra peak in ACM/w.
- **LSLx / Michigan:** Michigan has an LSL pattern and belongs to the latter group, however its LSL pattern is followed by a unique late peak occurring in March through May 2021, centered in mid-April. Therefore, we refer to Michigan’s pattern as “LSLx”.

- **00L / prairie states:** Seven of the ten prairie or Great Plains states, states that experienced the Dust Bowl drought of the 1930s, saw no anomalous mortality whatsoever until late into the COVID-era, until the fall of 2021. Here, cvp1 and smp1 are essentially zero or near-zero, and the only large feature is cvp2 ("00L" pattern). Easterly neighbouring states of Iowa, Missouri and Wisconsin also have this 00L pattern: Iowa, Kansas, Missouri, Montana, Nebraska, North Dakota, Oklahoma, South Dakota, and Wisconsin. The prairie states of New Mexico and Wyoming have a similar pattern, 0SL; whereas Texas has 0LL, and Colorado has LSL.
- **0SL / Central-Westerly and Central-Easterly states:** The cluster of adjacent states of Arkansas, Idaho, Kentucky, North Carolina, Tennessee, West Virginia, Wyoming, Nevada and Utah, and the prairie state of New Mexico, exhibit a "0SL" pattern. The 00L and 0SL patterns are similar: in 00L we characterize smp1 as "near-zero", whereas in 0SL we characterize smp1 as "small".
- **0SL / North-Westerly coastal states:** The North-Westerly coastal states of Oregon and Washington also have the 0SL pattern; and a sharp (one-week) heatwave signal discussed below (section 3.4).
- **SBL / North-Easterly states:** Minnesota, New Hampshire, Ohio, and Virginia exhibit an "SBL" pattern, intermediate between SSL and S0L.
- **SSL / California and Georgia:** California and Georgia exhibit similar patterns to each other, in which both cvp1 and smp1 are distinct but small or medium, and cvp2 is very large. We refer to this as an "SSL" pattern. The SSL pattern occurs in populous states but is otherwise similar to the 00L and 0SL patterns, in that relatively small or near-zero excess mortality occurs until late into the COVID-era, until the fall of 2021 when cvp2 starts and becomes a large feature in ACM/w.
- **0LL / Southern states:** Both Florida and Texas exhibit a "0LL" pattern in cvp1-smp1-cvp2 in which cvp1 is essentially zero, whereas smp1 and cvp2 are both large. Most of the most southerly states exhibit this pattern: Alabama, Arizona, Florida, Mississippi, South Carolina, and Texas; whereas Louisiana exhibits a pattern in which all three features are large, an "LLL" pattern. Thus, the Southern

states are generally characterized and distinguished by large mortalities in the summer of 2020, which is exceptional for these states, followed by large mortalities in the fall and winter of 2020-2021.

- **LLL / Louisiana:** Louisiana is the only state that has all three main features in ACM/w (cvp1, smp1, cvp2) being comparable and large. It is the only Southern state that experienced a large cvp1 mortality at the start of the COVID-era.
- The remaining states, Vermont and Maine, have borderline patterns to those described above, which could be characterized as 00S and 0SS, respectively.
- The summer-2021 feature “smp2” occurs in virtually all the states (see Appendix).

This distribution of cvp1-smp1-cvp2 pattern type is shown, colour coded, on a map of the USA, in Figure 8.

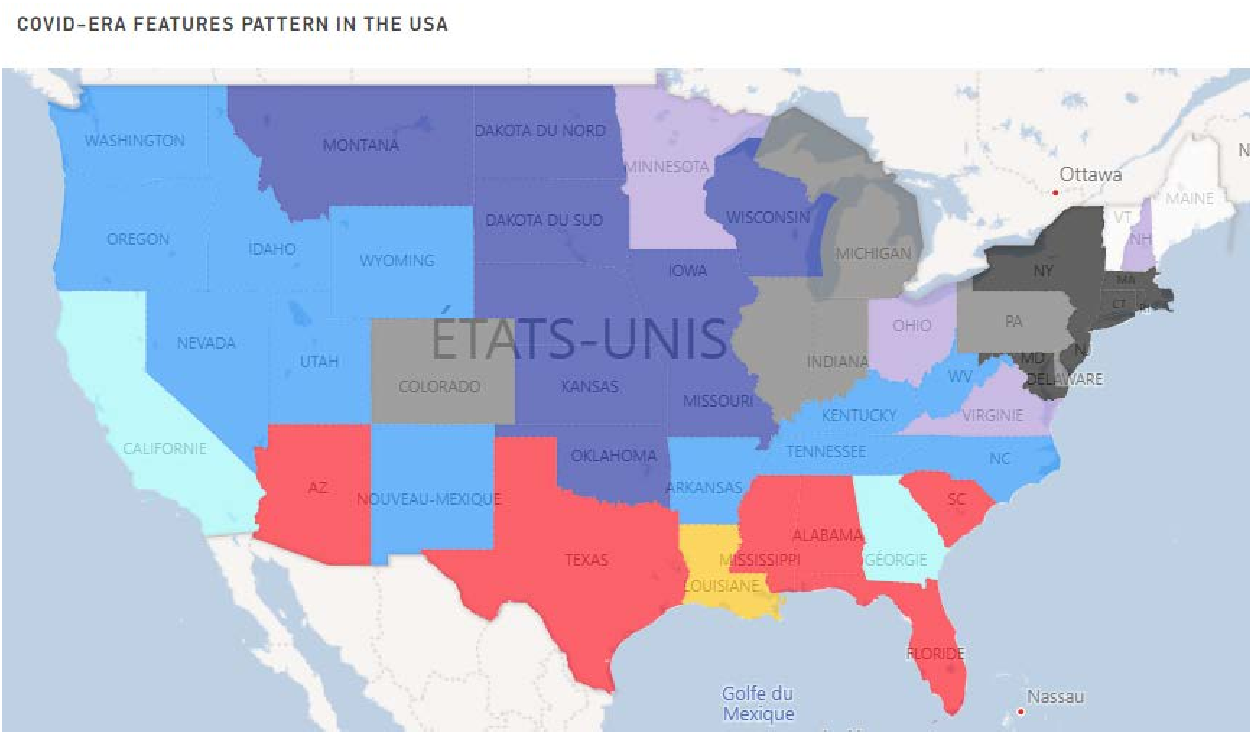


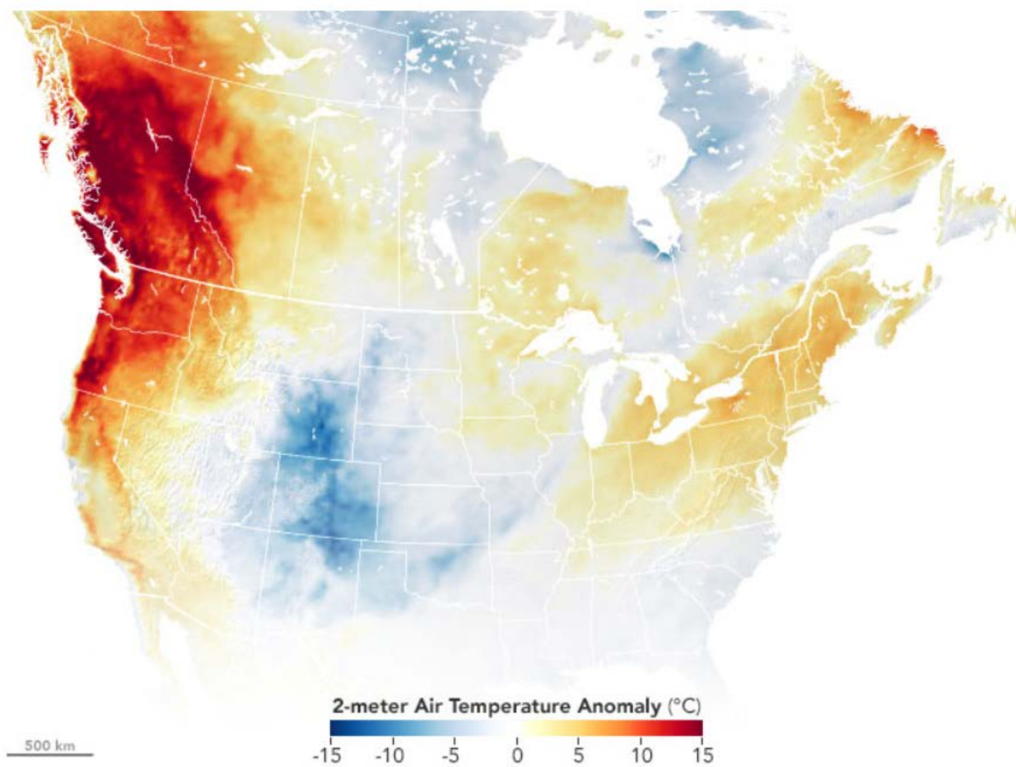
Figure 8. Map of COVID-era features pattern in the USA. The different colours represent the different pattern groups discussed in the text: black = L0M, gray = LSL, dark blue = 00L, blue = 0SL, light blue = SSL, purple = SBL, red = 0LL, yellow = LLL, white = 00S and 0SS. The first character of the pattern characterizes the cvp1 feature, the second the smp1 feature and the last the cvp2 feature. L stands for large, M for medium, S for small, B for borderline and 0 for zero / near-zero.

3.4. Late-June 2021 heatwave event in ACM/w for Oregon and Washington

There are sharp peaks (a single week or so) in the ACM/w data for Oregon and Washington, occurring at week-26 of 2021, which is the week of 28 June 2021 (Appendix).

The increased deaths coincide with an extraordinary weather event: The two states and British Columbia (Canada) experienced a short but record-breaking summer heatwave. NASA Earth Observatory (2021) described the heatwave as follows:

> The second map shows air temperature anomalies across the continental United States and Canada on June 27, 2021, when the heat intensified and records started to fall. The map is derived from the Goddard Earth Observing System (GEOS) model and depicts air temperatures at 2 meters (about 6.5 feet) above the ground. Red areas are where air temperatures climbed more than 27°F (15°C) higher than the 2014-2020 average for the same day.



Taking peak-to-local-baseline values, we estimate excess deaths from the heatwave to have been 246 and 475 deaths, respectively for Oregon and Washington.

This is a reminder of the deadliness of stress from atmospheric heat, which is relevant to our discussion about the COVID-era anomalies in the USA (below). We previously quantified such a heat-wave mortality event that occurred in France in 2003 (Rancourt et al., 2020).

3.5. ACM-SB/w normalized by population (ACM-SB/w/pop), by state

The different state-wise patterns of mortality in the USA during the COVID-era are best examined using ACM-SB/w normalized by population, ACM-SB/w/pop, and by reference to the cvp1-smp1-cvp2 patterns identified above. Normalization by population allows direct comparisons of the data for states with different populations.

In the following figures, normalization was done as follows:

Normalization of a cycle-year N was done with the population estimated just before the start of the cycle-year. Population estimates are each year on July 1st. The cycle-year starts on week-31 of a calendar-year (beginning of August). At the date of access, population estimates were from 2010 to 2020, so the cycle-year 2022 (last weeks of the data set) was normalized by the last available population estimate, the one for 2020.

When at the state level, the population used for normalization is the population of the specific state.

ACM-SB/w/pop curves are shown by groups of similar behaviours in Figure 9, as:

(a) L0M / North-Easterly coastal states: Connecticut, Maryland, Massachusetts, New Jersey, and New York.
(b) LSL / North-Central-Easterly non-coastal states: Colorado, Illinois, Indiana, Michigan (LSLx), and Pennsylvania.

(c) 00L / prairie states: Iowa, Kansas, Missouri, Montana, Nebraska, North Dakota, Oklahoma, and South Dakota. (Wisconsin is excluded because of bad data points for 2015, see Appendix.)
(d) 0SL / Central-Westerly non-coastal states: Idaho, Nevada, New Mexico, Utah, Wyoming.
(e) 0SL / North-Westerly coastal states: Oregon and Washington. (With June-2021 heatwave peak.)
(f) SSL / California and Georgia: California and Georgia.
(g) 0LL / Southern states: Arizona, Florida, Mississippi, South Carolina, and Texas (Alabama is excluded because of bad data points for 2014, see Appendix).
(h) LLL / Louisiana: Louisiana, shown with Michigan.

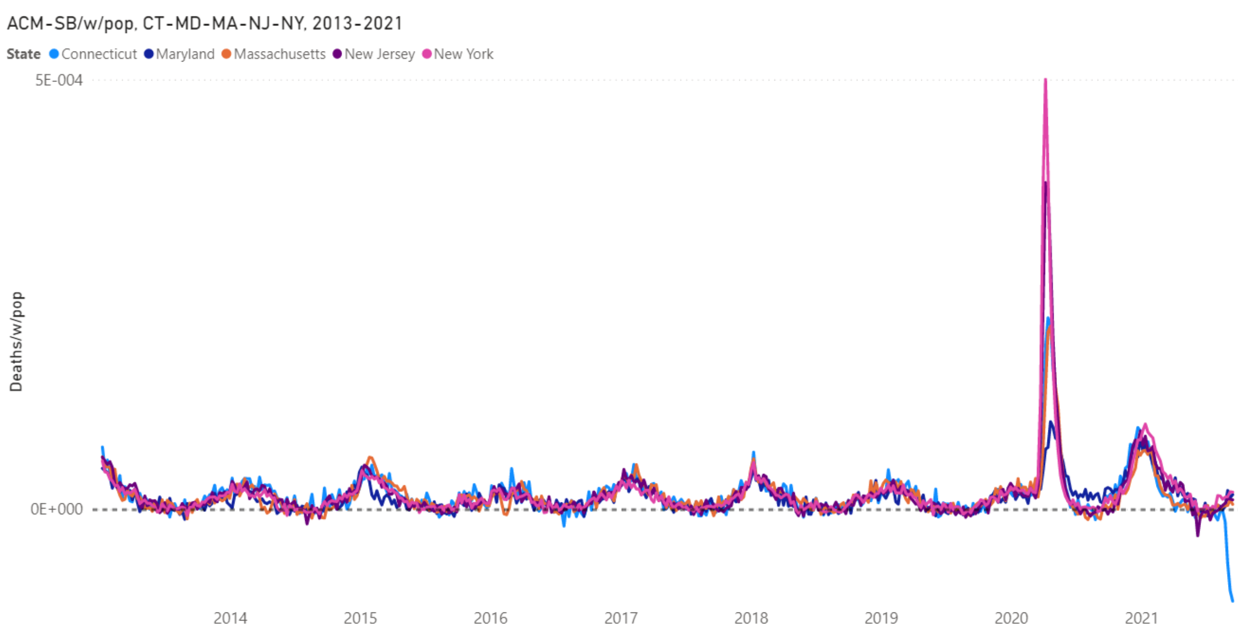


Figure 9a. Difference between all-cause mortality and summer baseline mortality by week normalized by population for Connecticut, Maryland, Massachusetts, New Jersey and New York from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

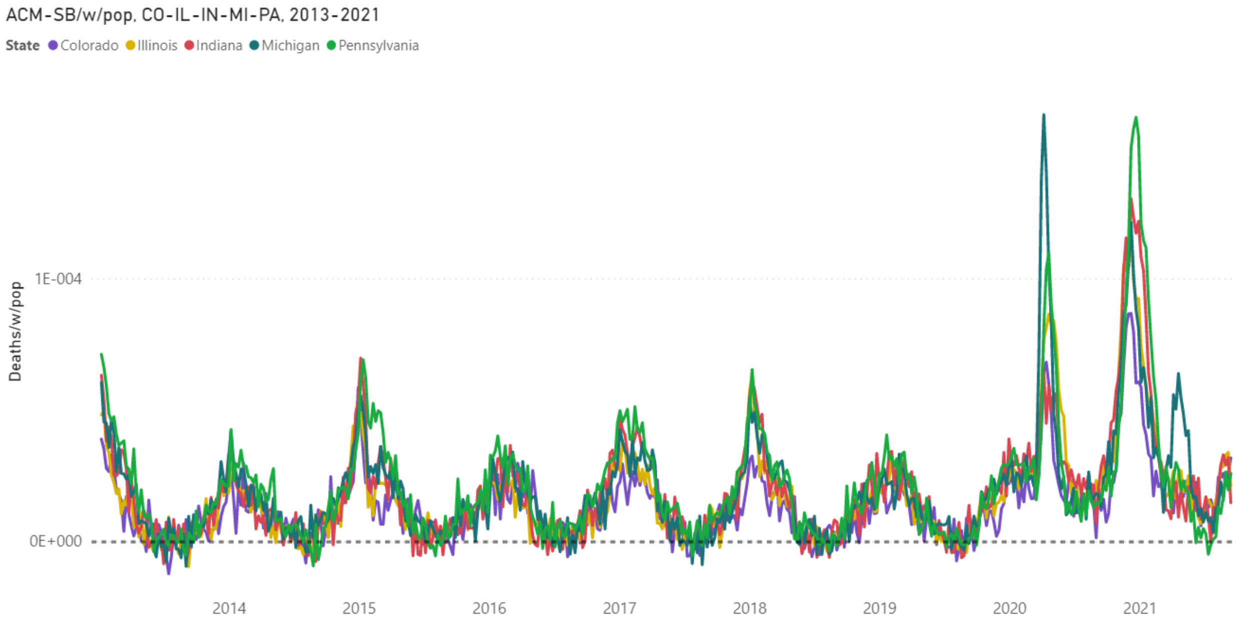


Figure 9b(i). Difference between all-cause mortality and summer baseline mortality by week normalized by population for Colorado, Illinois, Indiana, Michigan and Pennsylvania from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

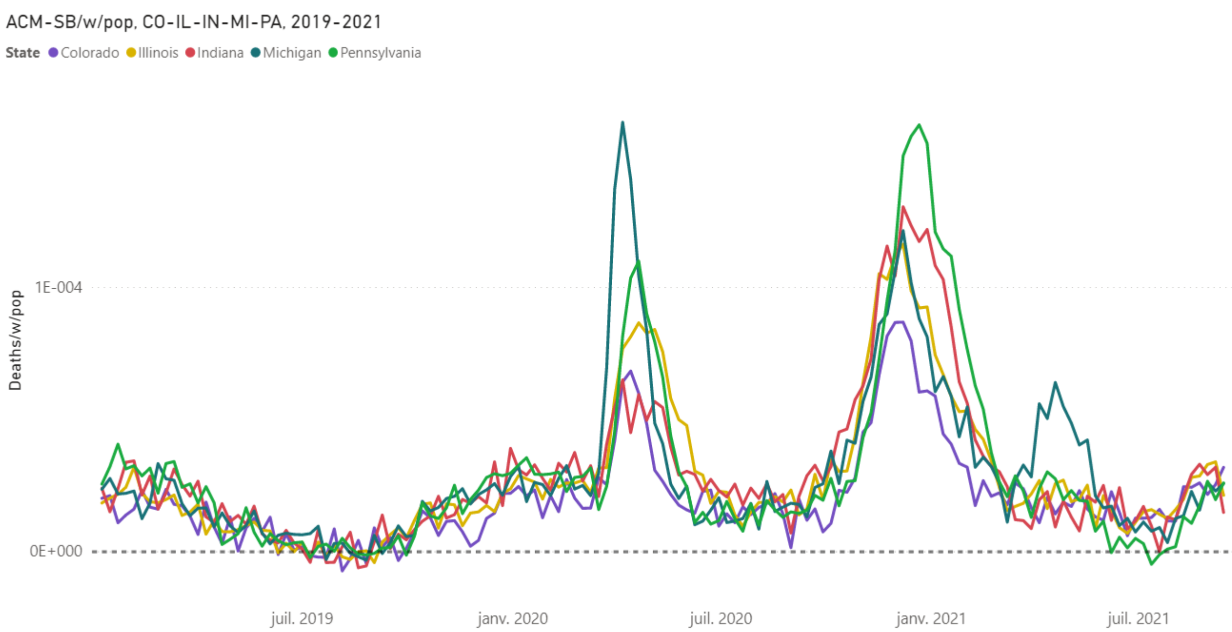


Figure 9b(ii). Difference between all-cause mortality and summer baseline mortality by week normalized by population for Colorado, Illinois, Indiana, Michigan and Pennsylvania from 2019 to 2021. Data are displayed from week-1 of 2019 to week-37 of 2021. The dashed

line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

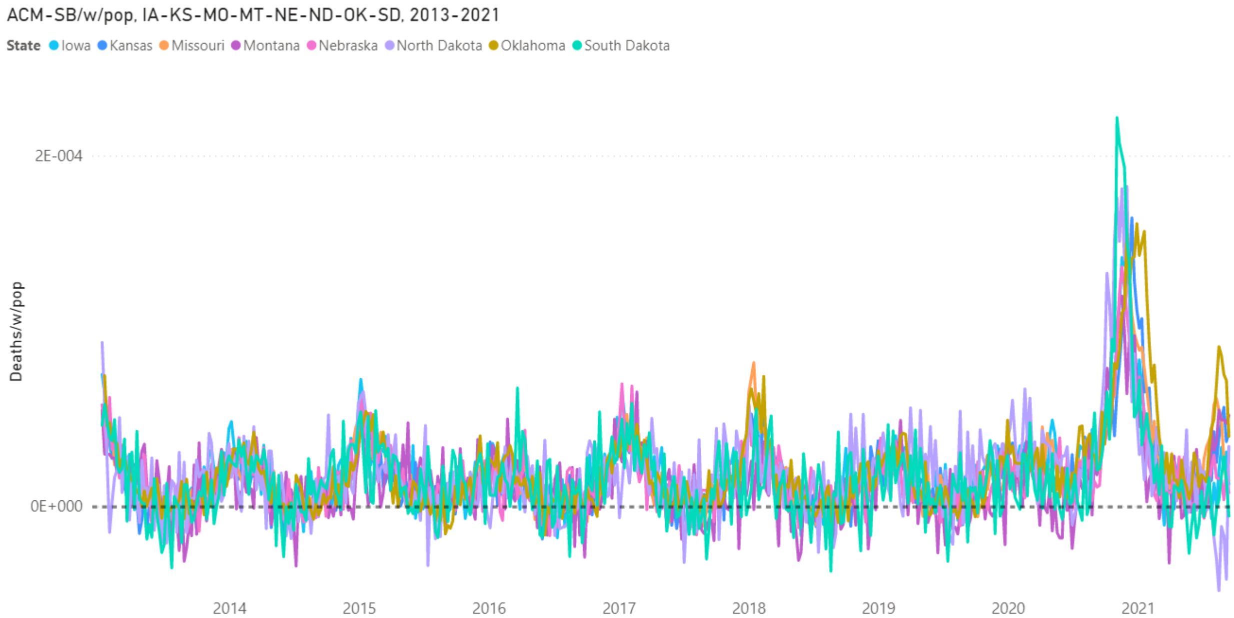


Figure 9c. Difference between all-cause mortality and summer baseline mortality by week normalized by population for Iowa, Kansas, Missouri, Montana, Nebraska, North Dakota, Oklahoma and South Dakota from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

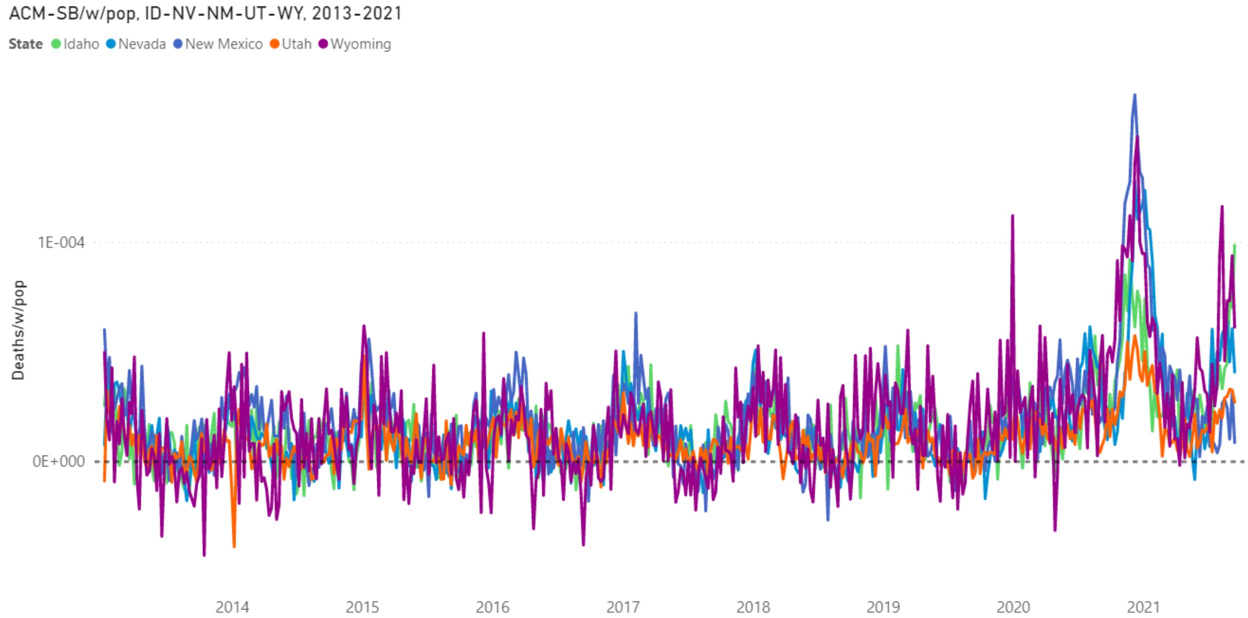


Figure 9d. Difference between all-cause mortality and summer baseline mortality by week normalized by population for Idaho, Nevada, New Mexico, Utah and Wyoming from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

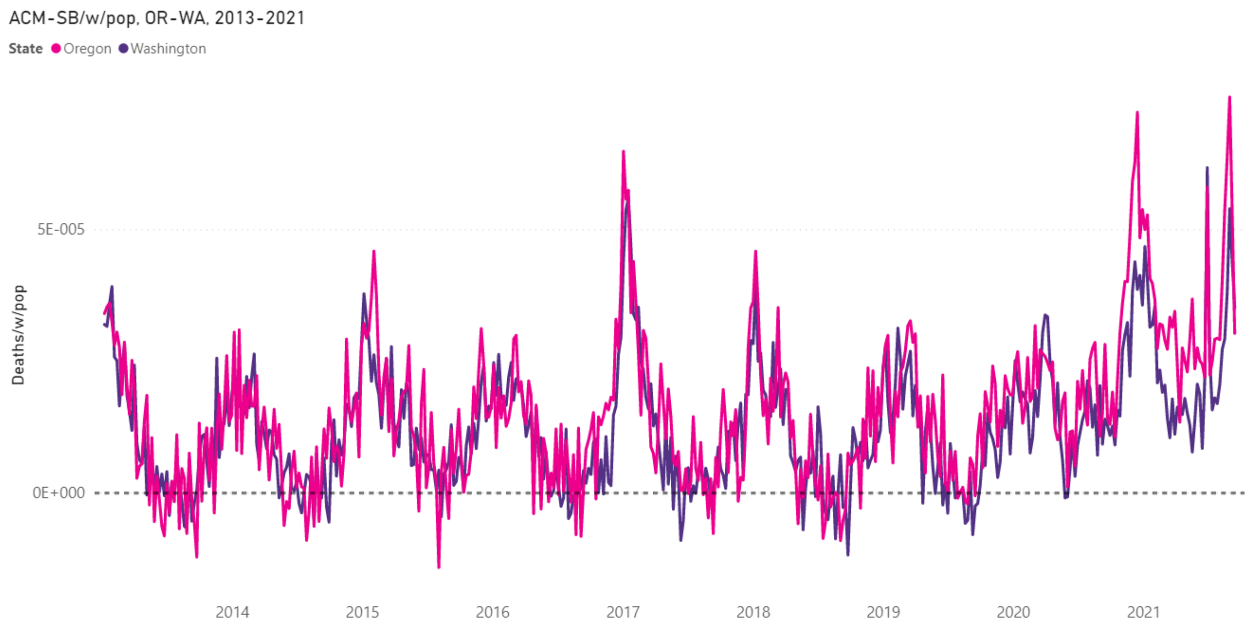


Figure 9e. Difference between all-cause mortality and summer baseline mortality by week normalized by population for Oregon and Washington from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM

data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

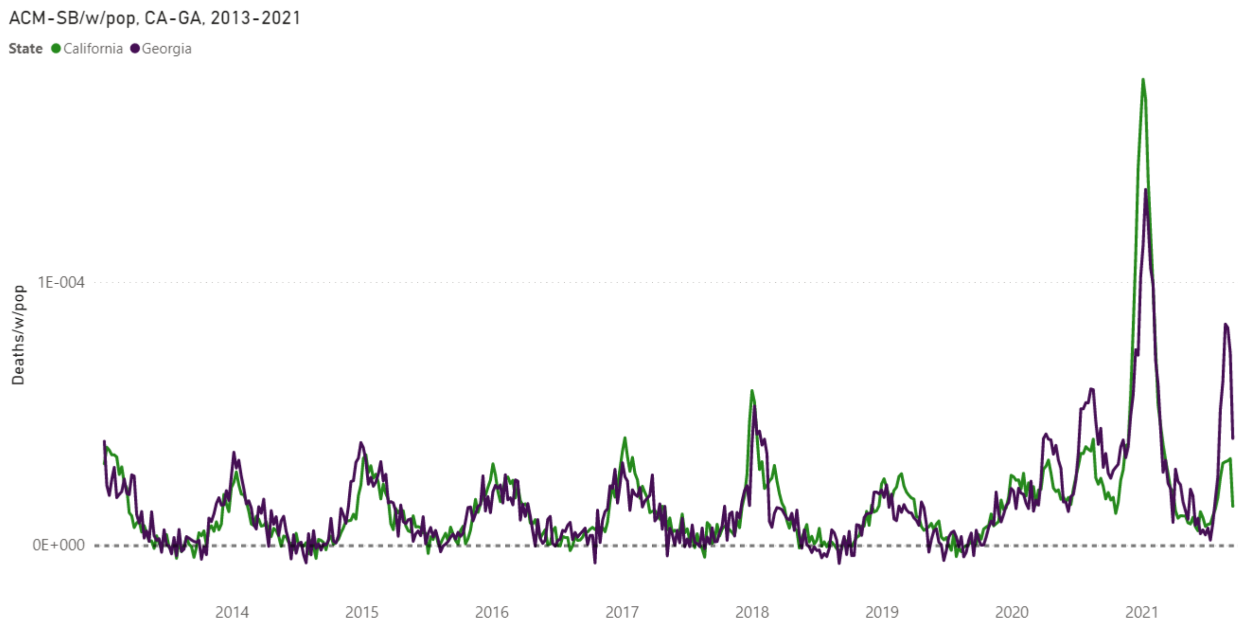


Figure 9f. Difference between all-cause mortality and summer baseline mortality by week normalized by population for California and Georgia from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

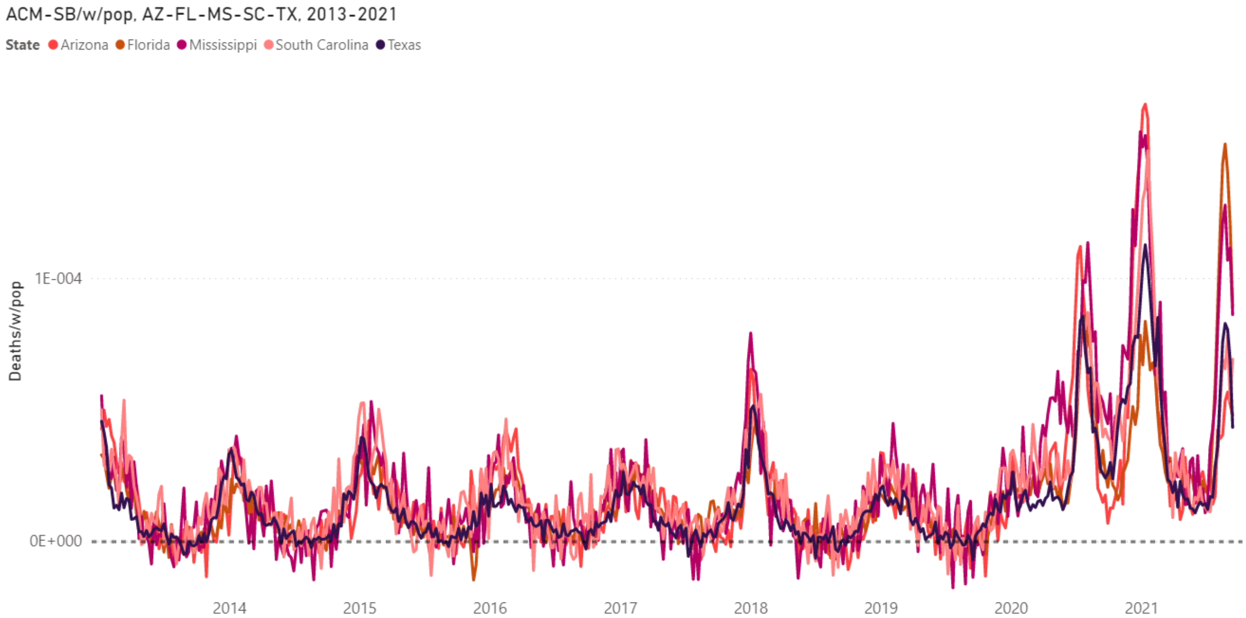


Figure 9g. Difference between all-cause mortality and summer baseline mortality by week normalized by population for Arizona, Florida, Mississippi, South Carolina and Texas from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

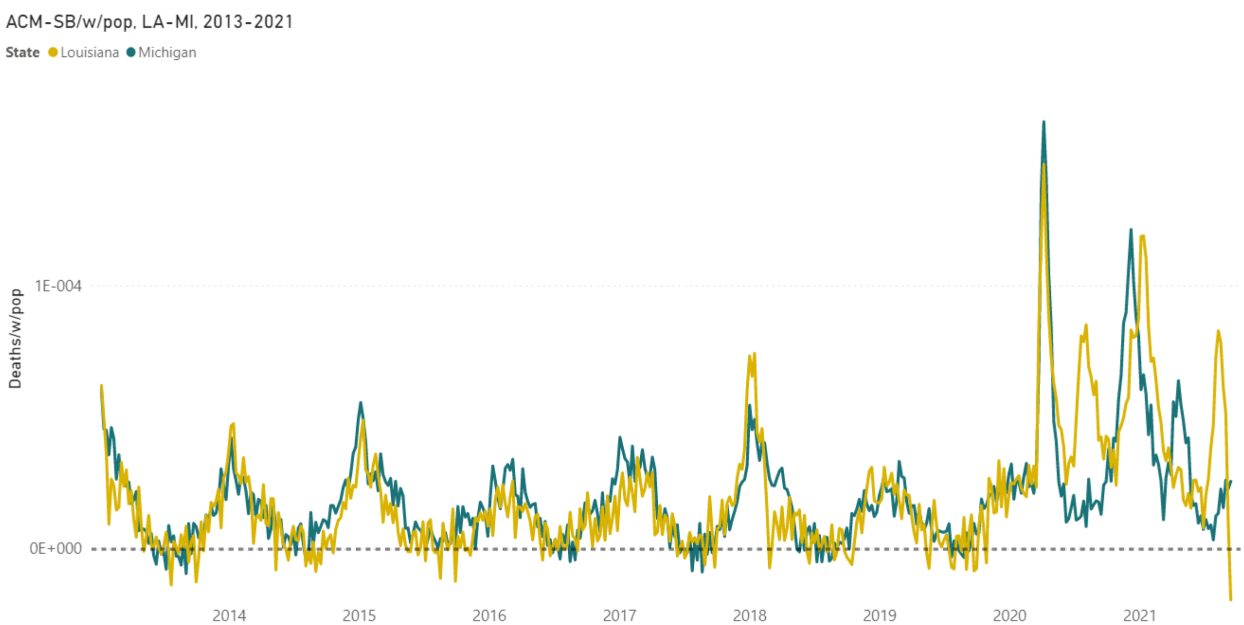


Figure 9h(i). Difference between all-cause mortality and summer baseline mortality by week normalized by population for Louisiana and Michigan from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM

data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

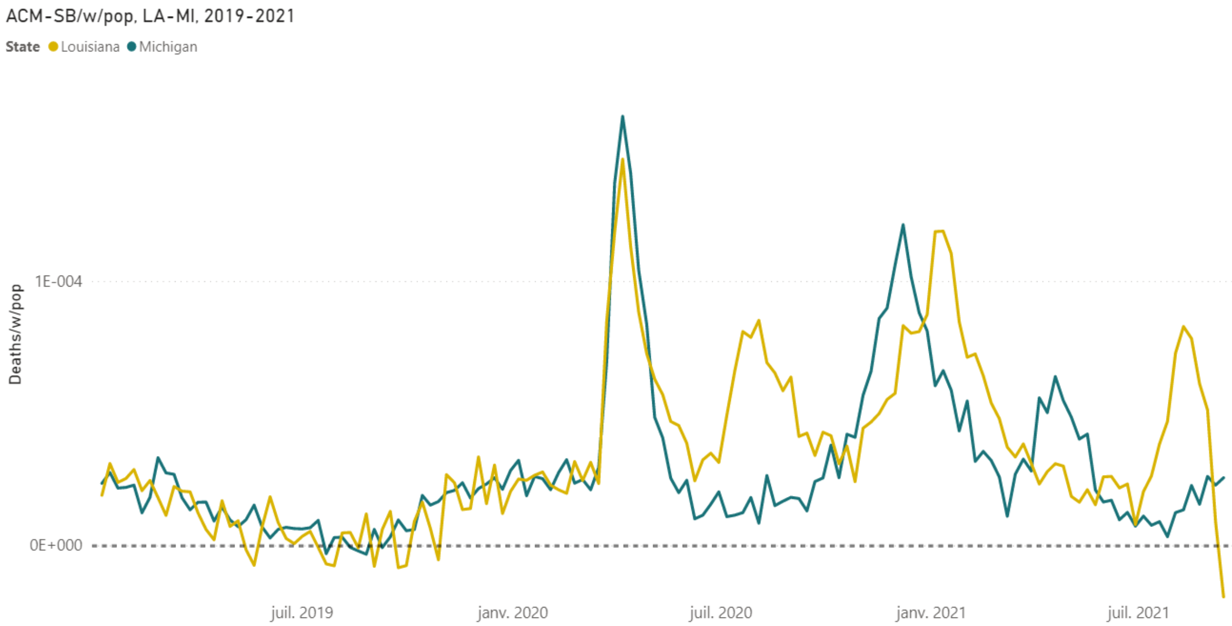


Figure 9h(ii). Difference between all-cause mortality and summer baseline mortality by week normalized by population for Louisiana and Michigan from 2019 to 2021. Data are displayed from week-1 of 2019 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

Figures 8 and 9 show that there are large state-to-state differences in COVID-era mortality by time, and that these differences approximately group into four (4) types, by geographical region, as:

- L0M : North-East coastal states
- LSL : North-East non-coastal states
- 00L / 0SL / SSL / SBL : Central and Western-Eastern states
- 0LL : Southern states

Louisiana is unique, with an LLL pattern, and large mortality in all three periods (cvp1, smp1, cvp2). Michigan (LSLx) has a unique late peak, occurring in March through May

2021, centered on mid-April 2021. Oregon and Washington have unique June-2021 single-week heatwave peaks.

This description is “coarse grain” and is simplified. For example, California has a distinct cvp1 feature even though it is much smaller than that occurring in the North-East states. Also, what happened in New York City is literally off-the-charts regarding cvp1 (Rancourt, 2020).

A most striking aspect of mortality during the COVID-era is precisely the state-wise heterogeneity in ACM by time, which we have described and illustrated above, and in the Appendix. This is striking because the seasonal cycle of all-cause deaths is usually remarkably uniform from state to state, from country to country, from province to province, from county to county… through all the inferred and declared epidemics and pandemics of viral respiratory diseases. Although the shapes of ACM by time change from season to season, the shapes for a given year are nonetheless synchronous and essentially the same across regions, over a global hemisphere, since good data has been available, since the end of the Second World War in most Western countries (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021).

Indeed, as an aside, we consider that this empirical fact (geographic homogeneity of synchronous mortality by time curves) represents a hard challenge against the theory that viral respiratory diseases spread person-to-person by proximity or “contact” and that such spread drives epidemics and pandemics, at the population level.

We quantify the said geographical heterogeneity of the COVID-era mortality by time below, but first we illustrate it further with direct comparisons of the ACM-SB/w/pop curves for states in different regions, with different cvp1-smp1-cvp2 patterns.

Figure 10 shows ACM-SB/w/pop for one state from each of the following cvp1-smp1-cvp2 patterns: California (SSL), Florida (0LL), Michigan (LSLx), Nevada (0SL), New York (L0M), South Dakoda (00L).

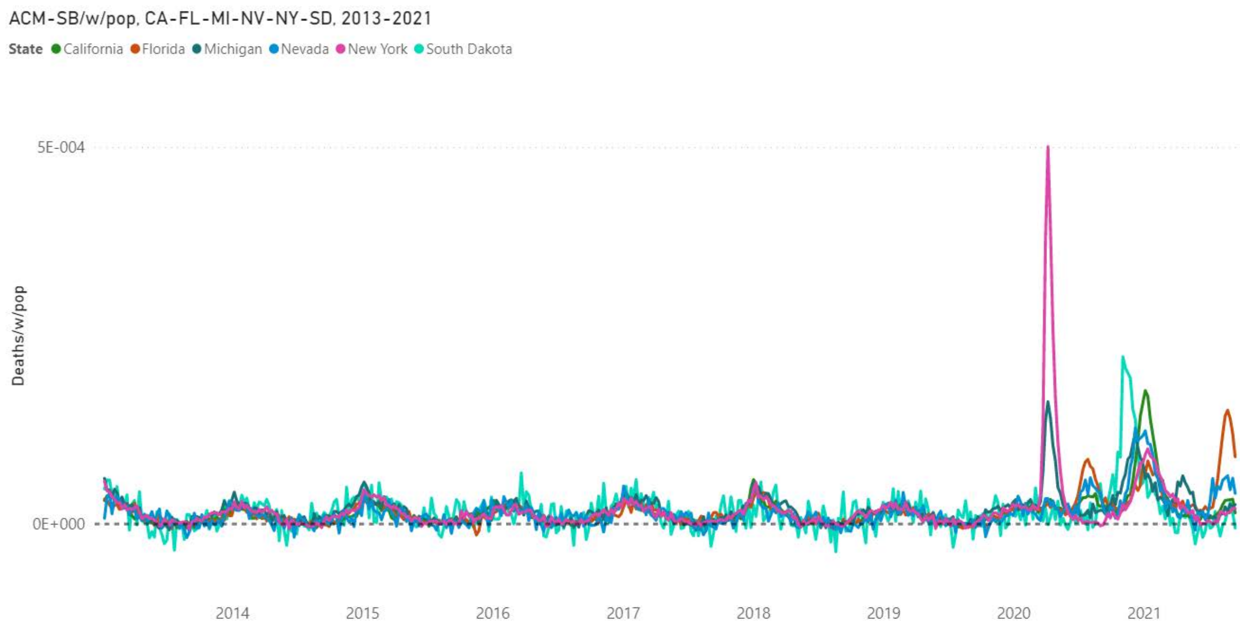


Figure 10a. Difference between all-cause mortality and summer baseline mortality by week normalized by population for California, Florida, Michigan, Nevada, New York and South Dakota from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

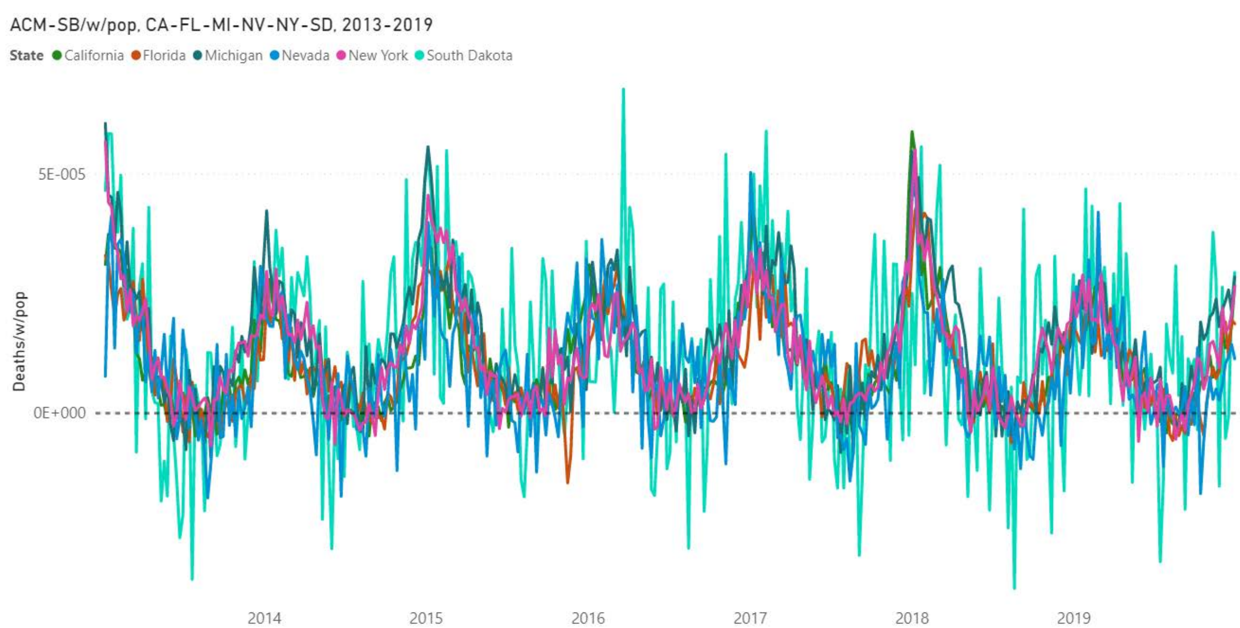


Figure 10b. Difference between all-cause mortality and summer baseline mortality by week normalized by population for California, Florida, Michigan, Nevada, New York and South Dakota from 2013 to 2019. Data are displayed from week-1 of 2013 to week-52 of 2019.

The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

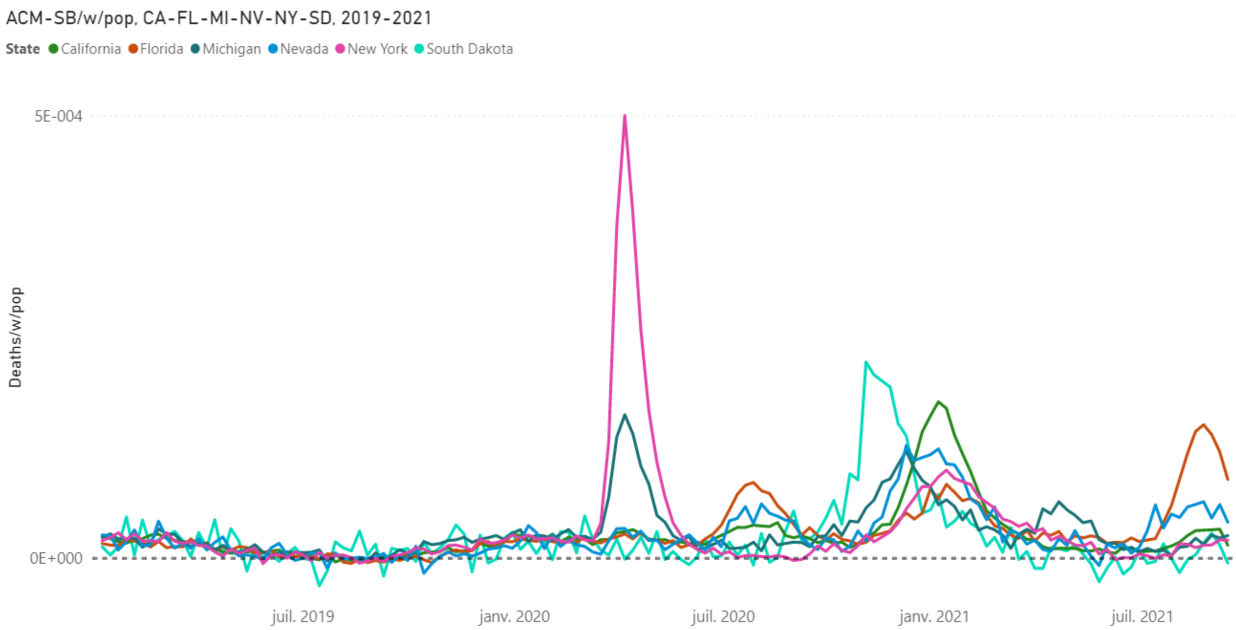


Figure 10c. Difference between all-cause mortality and summer baseline mortality by week normalized by population for California, Florida, Michigan, Nevada, New York and South Dakota from 2019 to 2021. Data are displayed from week-1 of 2019 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

Figure 11 makes the same kind of comparison for states that have large cvp1 features: Colorado (LSL), Connecticut (L0M), Illinois (LSL), Louisiana (LLL), New Jersey (L0M), New York (L0M).

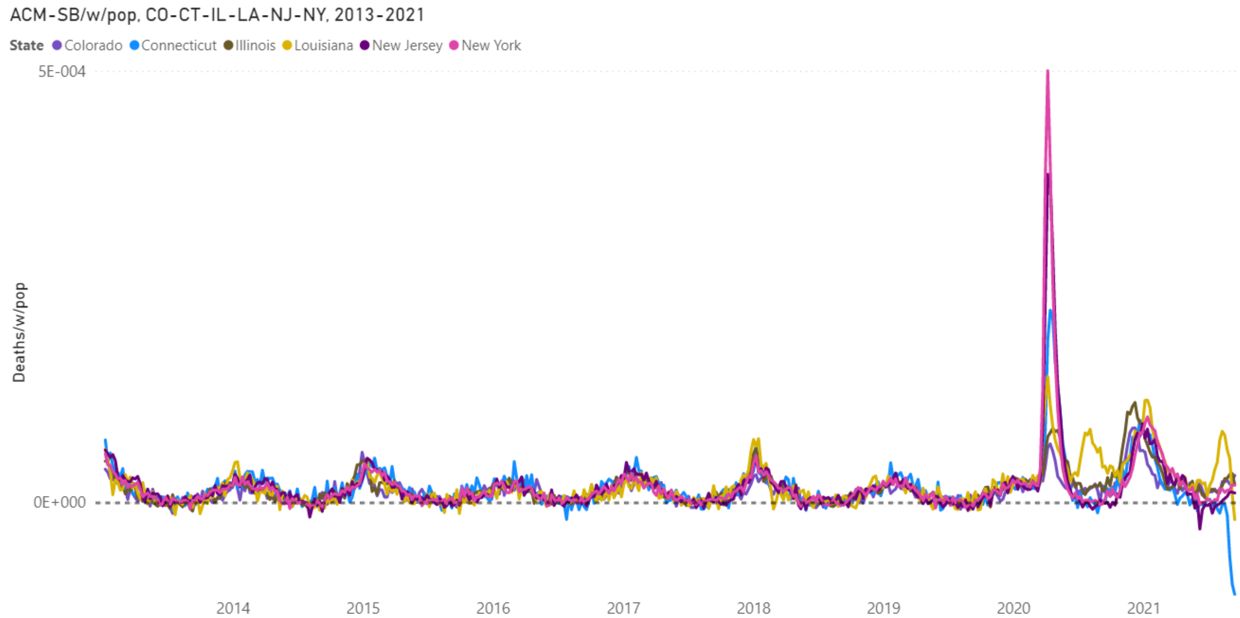


Figure 11a. Difference between all-cause mortality and summer baseline mortality by week normalized by population for Colorado, Connecticut, Illinois, Louisiana, New Jersey and New York from 2013 to 2021. Data are displayed from week-1 of 2013 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

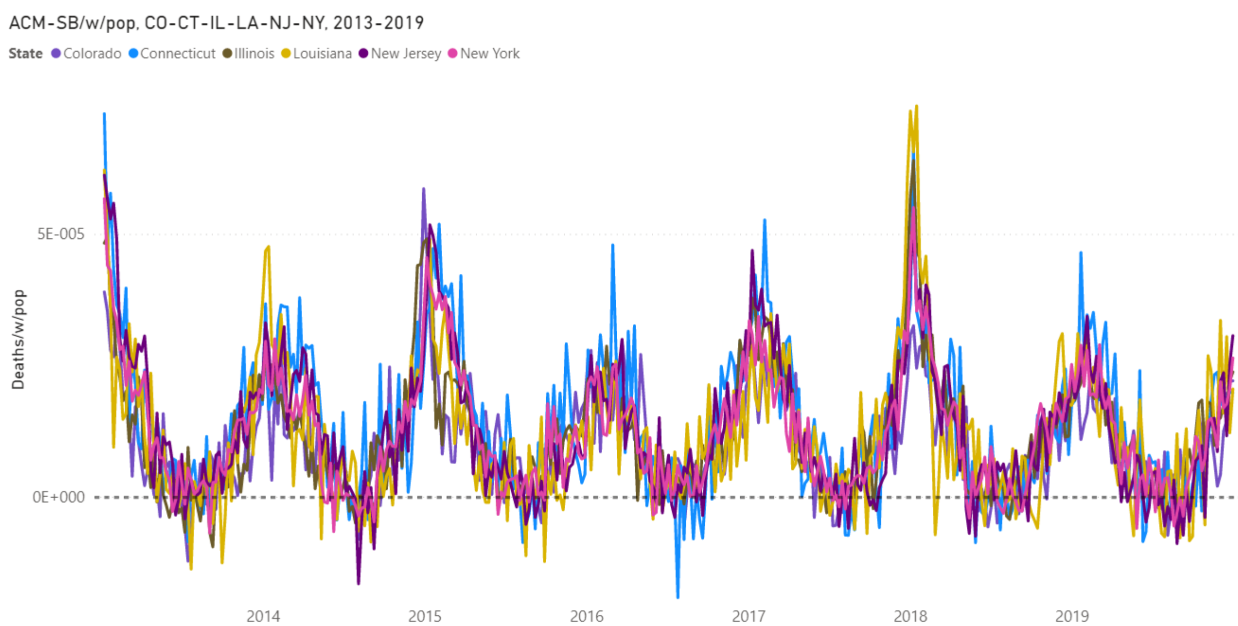


Figure 11b. Difference between all-cause mortality and summer baseline mortality by week normalized by population for Colorado, Connecticut, Illinois, Louisiana, New Jersey and New York from 2013 to 2019. Data are displayed from week-1 of 2013 to week-52 of

2019. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

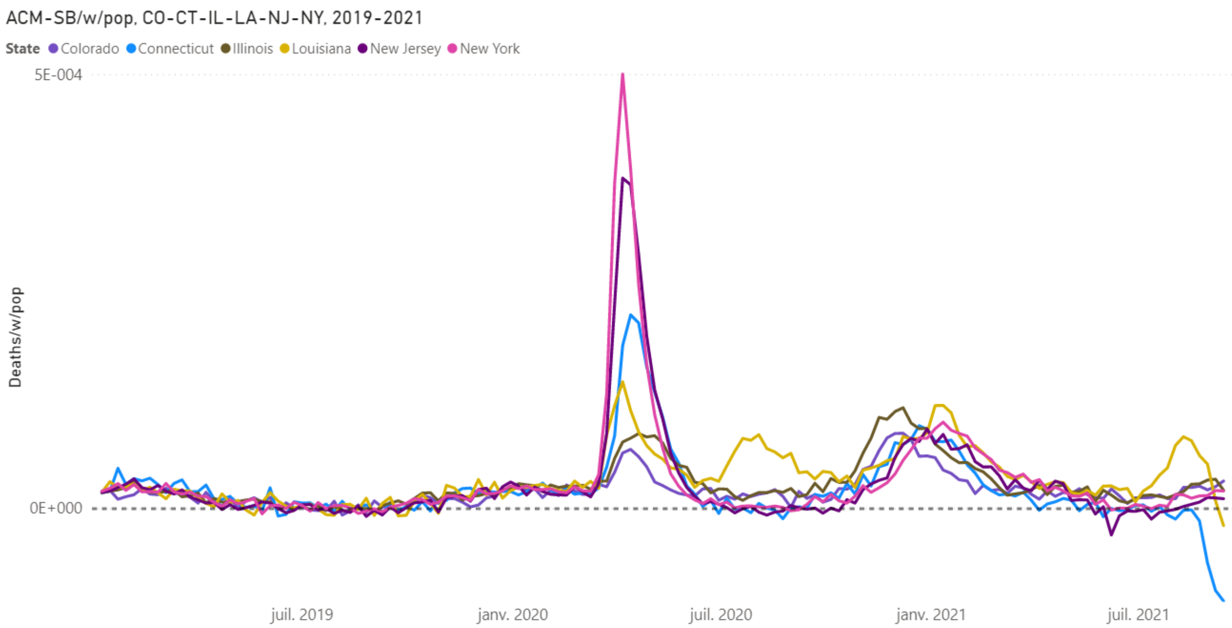


Figure 11c. Difference between all-cause mortality and summer baseline mortality by week normalized by population for Colorado, Connecticut, Illinois, Louisiana, New Jersey and New York from 2019 to 2021. Data are displayed from week-1 of 2019 to week-37 of 2021. The dashed line emphasizes the zero. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

3.6. ACM-SB by cycle-year (winter burden, WB) by population (WB/pop), USA and state-to-state variations

Next, we analyse ACM-SB/w in terms of integrated intensities over cycle-years. By definition, the said integrated intensity is the "winter burden", WB, for the given cycle-year. WB is the excess (above-SB) mortality per cycle-year. We normalize WB by population, WB/pop, in order to make state-to-state and state-to-nation comparisons.

Figure 12a shows the WB/pop, for cycle-years 2014 to 2021 (cycle-year 2021 contains and is approximately centered on January 2021, and so on), for the entire continental

USA (49 states). We see the seasonal (year to year) variations 2014-2019, followed by the large COVID-era increase 2020-2021, which echoes the large 2020 calendar-year increase shown in Figures 1 and 4.

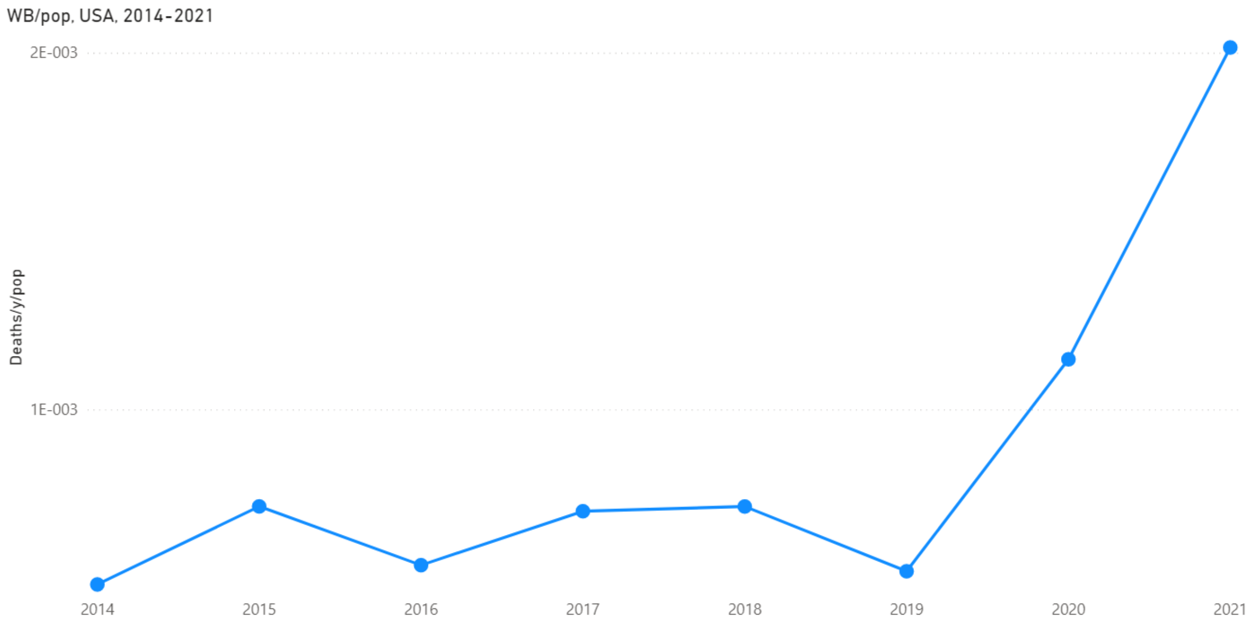


Figure 12a. Winter burden normalized by population in the USA for cycle-years 2014 to 2021. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated and WB calculated as described in section 2.

Figure 12b shows WB/pop versus cycle-year (2014-2021), for all the continental USA states on the same graph.

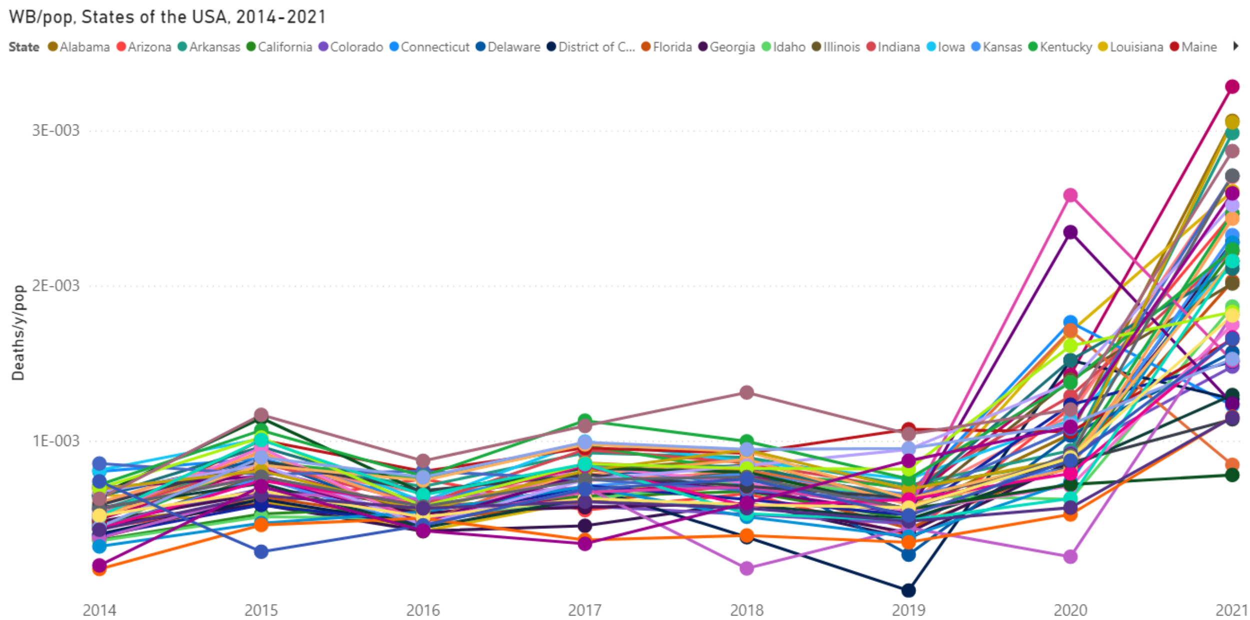


Figure 12b. Winter burden normalized by population for each of the continental states of the USA for cycle-years 2014 to 2021. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). The 49 continental states include the District of Columbia and exclude Alaska and Hawaii. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated and WB calculated as described in section 2.

Figure 12c shows WB/pop versus cycle-year (2014-2021) for the "0LL" group of Southern states (having a cvp1-smp1-cvp2 0LL pattern), and for Louisiana, which has the cvp1-smp1-cvp2 "LLL" pattern, on the same graph. We note a larger 2020 WB/pop value for Louisiana, than would be expected for a Southern state, because its large LLL-pattern cvp1 feature increases its 2020 WB/pop value.

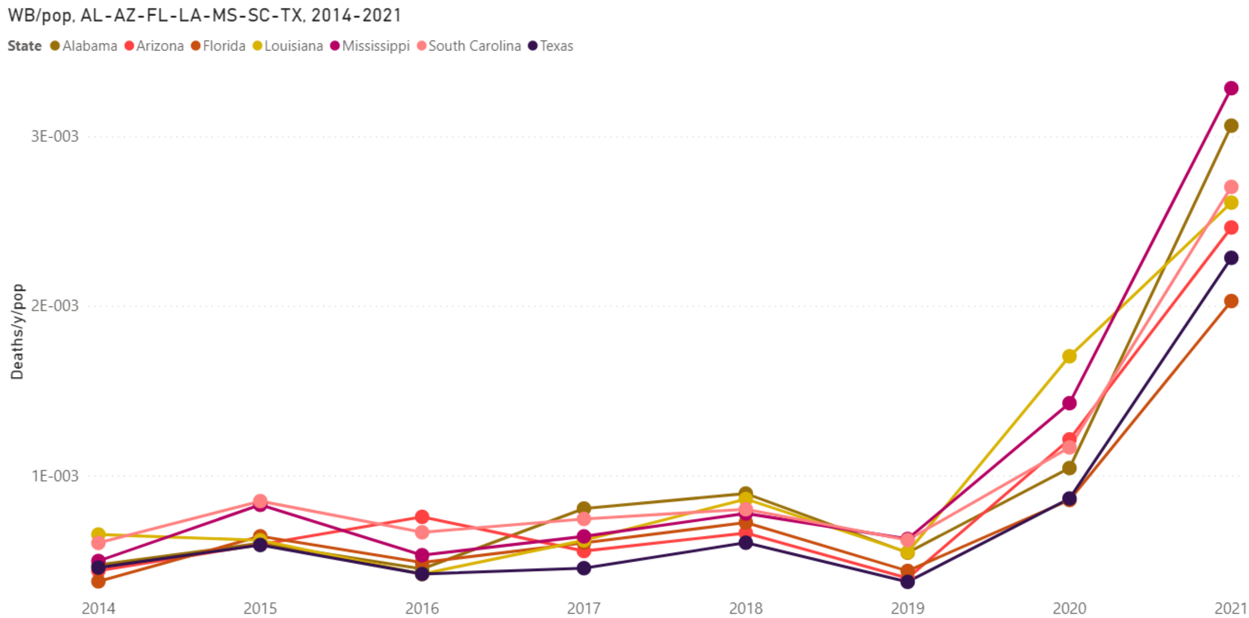


Figure 12c. Winter burden normalized by population in Alabama, Arizona, Florida, Louisiana, Mississippi, South Carolina and Texas for cycle-years 2014 to 2021. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated and WB calculated as described in section 2.

Figure 12d shows WB/pop versus cycle-year (2014-2021) for the “L0M” group of North-East coastal states (having a cvp1-smp1-cvp2 L0M pattern), including Maryland, which has a limit behaviour to be included in this group. Since this group has exceptionally large cvp1 features, we see that generally the WB-2020 is larger than the WB-2021.

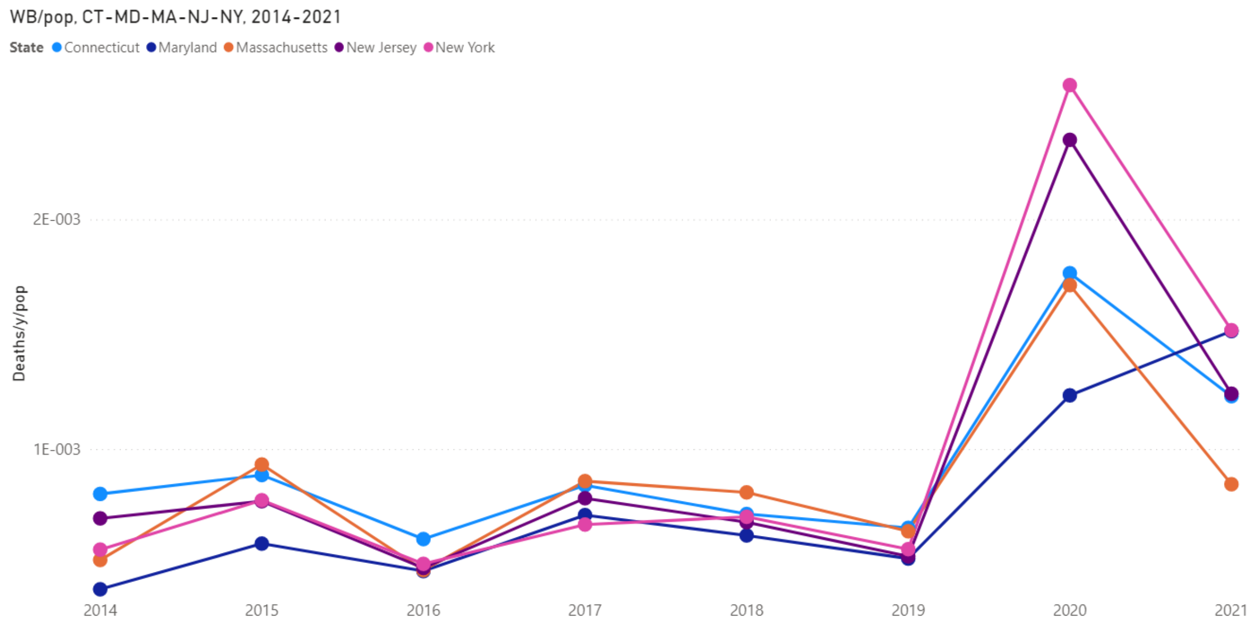


Figure 12d. Winter burden normalized by population in Connecticut, Maryland, Massachusetts, New Jersey and New York for cycle-years 2014 to 2021. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated and WB calculated as described in section 2.

Figure 12b shows that, like the ACM-SB/w/pop curves themselves would suggest (Figures 10 and 11), the state-to-state spread in WB/pop values is much larger in the COVID-era than in the previous decade or so. We can illustrate this pre-COVID/COVID-era difference by plotting the frequency distribution of state-to-state values of WB/pop for each cycle-year. These distributions are shown together in Figure 13.

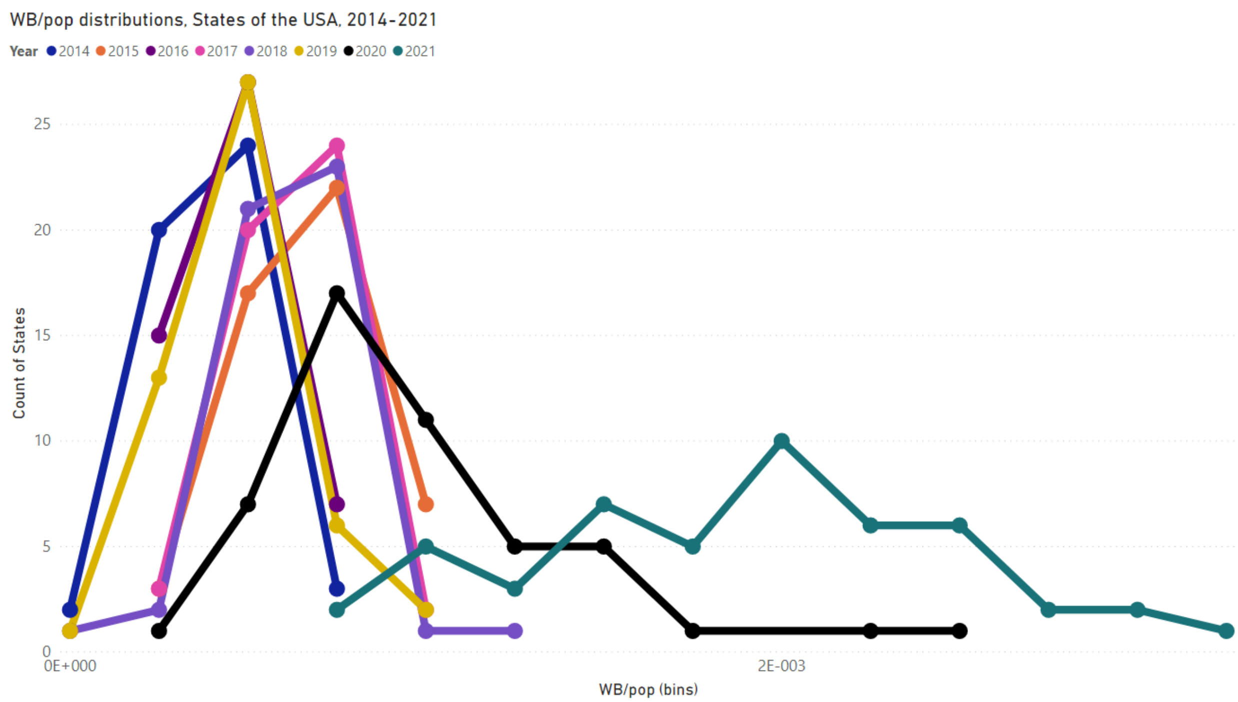


Figure 13. Frequency distributions of state-to-state values of WB/pop for each cycle-year, 2014-2021, as indicated by the colour scheme. Each distribution is normalized to 49, the number of continental USA states (including District of Columbia, excluding Alaska and Hawaii). A bin-width of 2.5E−4 deaths/pop was used. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated and WB calculated as described in section 2.

Here (Figure 13), it is interesting to note that the six pre-COVID-era cycle-years (2014-2019) fall into two distinct distribution types, with the same widths but positions differing by a set amount, corresponding to “light” (2014, 2016, 2019; less deadly winter) and “heavy” (2015, 2017, 2018; deadlier winter) years that are also recognized in the ACM/w or ACM-SB/w patterns themselves (e.g., Figures 5 and 6).

By comparison, the distribution for cycle-year 2020 has larger WB/pop values and a tail that extends far towards even larger values. The distribution for cycle-year 2021 is exceedingly wide and extends to extremely large values.

Properties of the frequency distributions (Figure 13) can be quantified as follows. For each distribution (for a given cycle-year) we calculate: the average ("av"), the median ("med"), the standard deviation ("sd"), and the difference "av-med". The latter difference av-med is related to the magnitude of the asymmetry of the distribution, and its sign indicates whether any extended tail extends toward small (negative) or large (positive) WB/pop values. These four parameters (av, med, sd, av-med) are shown versus cycle-year in Figure 14.

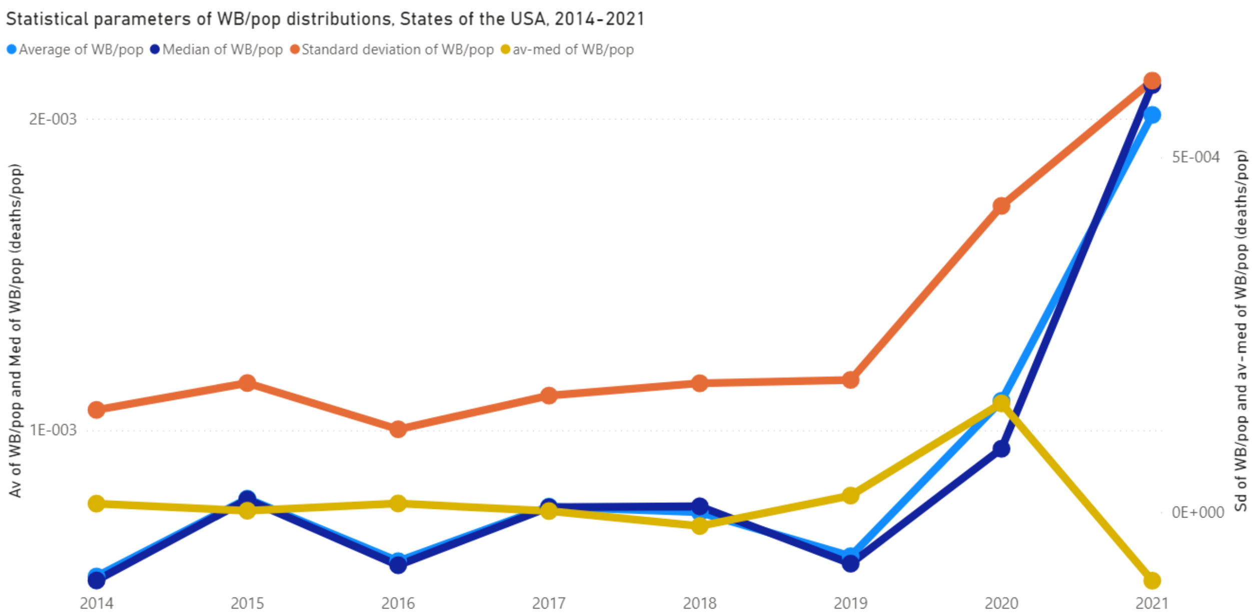


Figure 14. Statistical parameters of the WB/pop distributions of the 49 continental states of the USA for cycle-years 2014 to 2021. The 49 continental states include the District of Columbia and exclude Alaska and Hawaii. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated and WB calculated as described in section 2.

Here (Figure 14), the variations of "av" and "med" are generally those expected, given the behaviour of WB/pop versus cycle-year for the entire continental USA (Figure 12a).

The "sd" (Figure 14) has a remarkably constant pre-COVID-era (prior to 2020) value of approximately 1.6(1.2—1.9 range)E−4 deaths/pop, and then shoots up to 4.3E−4

(2020) and 6.1E−4 (2021) deaths/pop. In other words, the COVID-era is characterized by an anomalously large state-to-state heterogeneity in WB/pop values, an approximately 4-fold increase in absolute magnitude.

In fact, using WB/pop masks the actual state-wise heterogeneity, since the COVID-era features cvp1 and smp1 have a much larger intrinsic (relative) heterogeneity than WB. The said large heterogeneity is evident in the ACM-SB/w/pop data itself (Figures 10 and 11), but let us quantify it, and let us examine “asymmetry” (presence of tails) as well. We use the dimensionless parameters sd/av and (av-med)/av, which are as follows.

Breadth and asymmetry of state-wise distributions of integrated deaths		
feature	**sd/av**	**(av-med)/av**
pre-COVID-era WB/pop 2014-2019	0.20—0.31	-0.03—+0.04
2020 WB/pop	0.39	+0.14
cvp1/pop	0.79	+0.27
smp1/pop	0.67	+0.17
cvp2/pop	0.28	0.00
2021 WB/pop	0.30	-0.05

Table 2. Breadth and asymmetry of state-wise distributions of integrated deaths for the pre-COVID-era WB/pop, and for features in the COVID-era. Features in the COVID-era include 2020 WB/pop, cvp1/pop, smp1/pop, cvp2/pop and 2021 WB/pop.

The state-wise heterogeneity of cvp1 is massive (sd/av: 0.79 compared to ~0.25) ((av-med)/av: +0.27 compared to ~+0.01), since cvp1 consists of essentially one extreme region in the North-East coastal states. The state-wise heterogeneity of smp1 is large (sd/av: 0.67 compared to ~0.25) ((av-med)/av: +0.17 compared to ~+0.01), since smp1 consists of essentially an extreme region in the Southern states.

We have observed such COVID-era jurisdictional heterogeneity in many countries, and country-wise in Europe, and we have argued that it is contrary to pandemic behaviour, and contrary to any (1945-2021) season of viral respiratory disease burden in the Northern hemisphere, and arises mainly from jurisdictional differences in applied medical and government responses to the pronouncement of a pandemic (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021).

In contrast, cvp2, which is entirely within the 2021 cycle-year and is the cycle-year's main (winter) feature, has normal pre-COVID-era state-wise homogeneity (sd/av: 0.28 compared to 0.20—0.31) ((av-med)/av: 0.00 compared to -0.03—+0.04). This suggests that cvp2 is not affected by any widely different state-to-state applied responses, but rather is the result of a broad, sustained, and state-wise homogenous stress on the USA population.

3.7. Geographical distribution and correlations between COVID-era above-SB seasonal deaths: cvp1 (spring-2020), smp1 (summer-2020) and cvp2 (fall-winter-2020-2021)

Recall that Figure 7 shows how we integrate to obtain the total above-SB deaths in each of the operationally defined features cvp1, smp1 and cvp2. Since the peak positions are operationally the same for all states (barring the extra peak for Michigan), we use the same delimiting weeks throughout, those shown in Figure 7. We normalize the state-wise deaths by state-wise population, in order to allow state-to-state comparisons.

Figure 15 shows a map of cvp1/pop for the continental states of the USA.

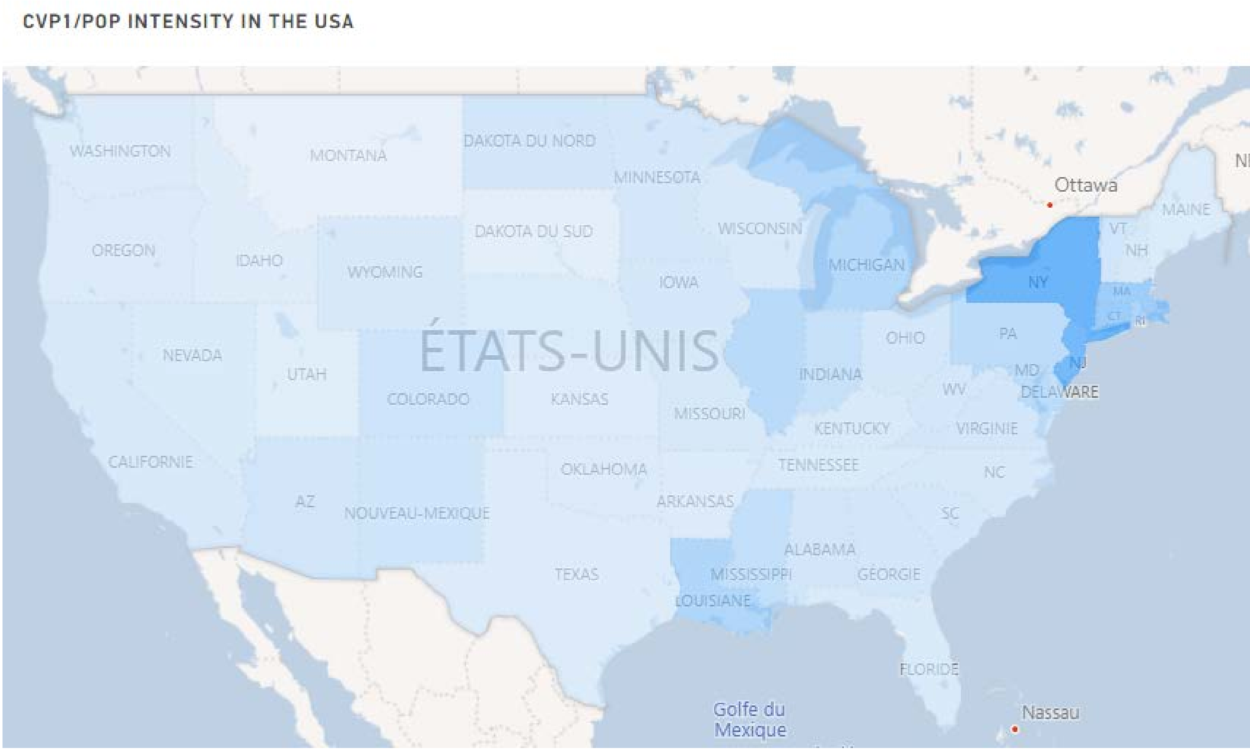


Figure 15. Map of the intensity of the cvp1 mortality normalized by population for the continental USA. Continental USA includes the District of Columbia and excludes Alaska and Hawaii. The cvp1 feature is the integrated deaths of ACM-SB between week-11 of 2020 and week-25 of 2020, inclusively. The darker the blue, the more intense the cvp1/pop. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

Here, we see that a cluster of North-East coastal states were essentially the only intense hot spot; and notable other states, including Louisiana, Illinois and Michigan, to a lesser degree. In fact, some 34 of the USA states do not have a resolved or detectable or significant cvp1 feature. We have described this previously (Rancourt, 2020) (Rancourt et al., 2020). We have argued that the cvp1 feature (the “covid peak”) is highly jurisdictionally heterogeneous, has a start synchronous with the 11 March 2020 WHO declaration of a pandemic, and is present throughout the mid-latitude Northern hemisphere, because it is caused by the medical and government responses to the declaration of a pandemic, especially in hospitals and care homes (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021). One can say with certainty that there

was no detectable or significant "first wave" in most of the USA, a phenomenon which is contrary to the very concept of a pandemic (Rancourt et al., 2021).

Figure 16 shows a map of smp1/pop for the continental states of the USA.

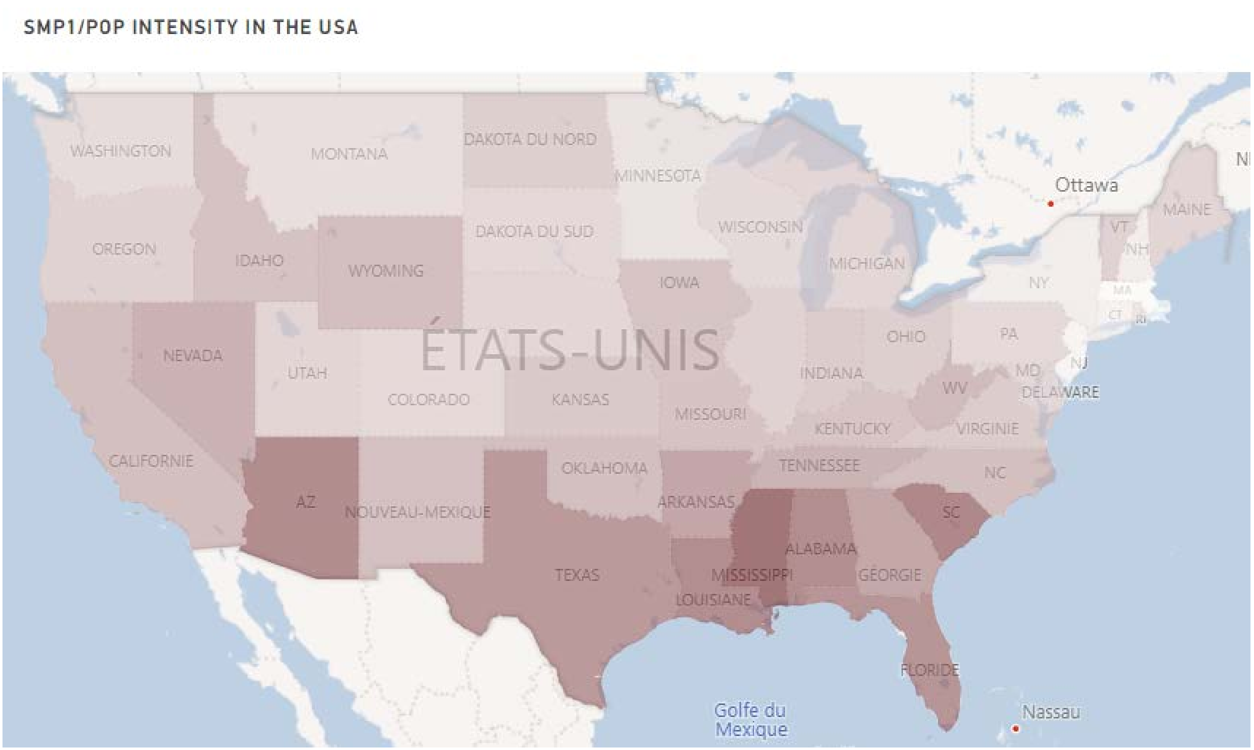


Figure 16. Map of the intensity of the smp1 mortality normalized by population for the continental USA. Continental USA includes the District of Columbia and excludes Alaska and Hawaii. The smp1 feature is the integrated deaths of ACM-SB between week-26 of 2020 and week-39 of 2020, inclusively. The darker the red, the more intense the smp1/pop. ACM data were retrieved from the CDC (CDC, 2021a) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021a), as described in Table 1. SB was estimated as described in section 2.

This is a remarkable map, which shows that the above-SB deaths in the summer of 2020 were concentrated in the Southern states of Arizona, Texas, Louisiana, Mississippi, Alabama, Florida and South Carolina. These results can be understood in terms of climatic, socio-economic and population health effects, as shown below. The results (Figure 16) are inconsistent with the theoretical concept of a viral respiratory disease pandemic. Furthermore, no previous large anomalous burden of all-cause

mortality has ever been concentrated in the Southern states, in one season, in the modern history of epidemiology for the USA.

There is no point showing a map of cvp2/pop for the continental states of the USA, because we showed above that the state-wise distribution of cvp2/pop is essentially homogeneous (Table 2). A map of cvp2/pop does not show any recognizable pattern.

Next, we examine whether there are any correlations or anti-correlations between the outcomes cvp1, smp1 and cvp2; and also smp2. Plots of one versus the other are as follows, in Figure 17.

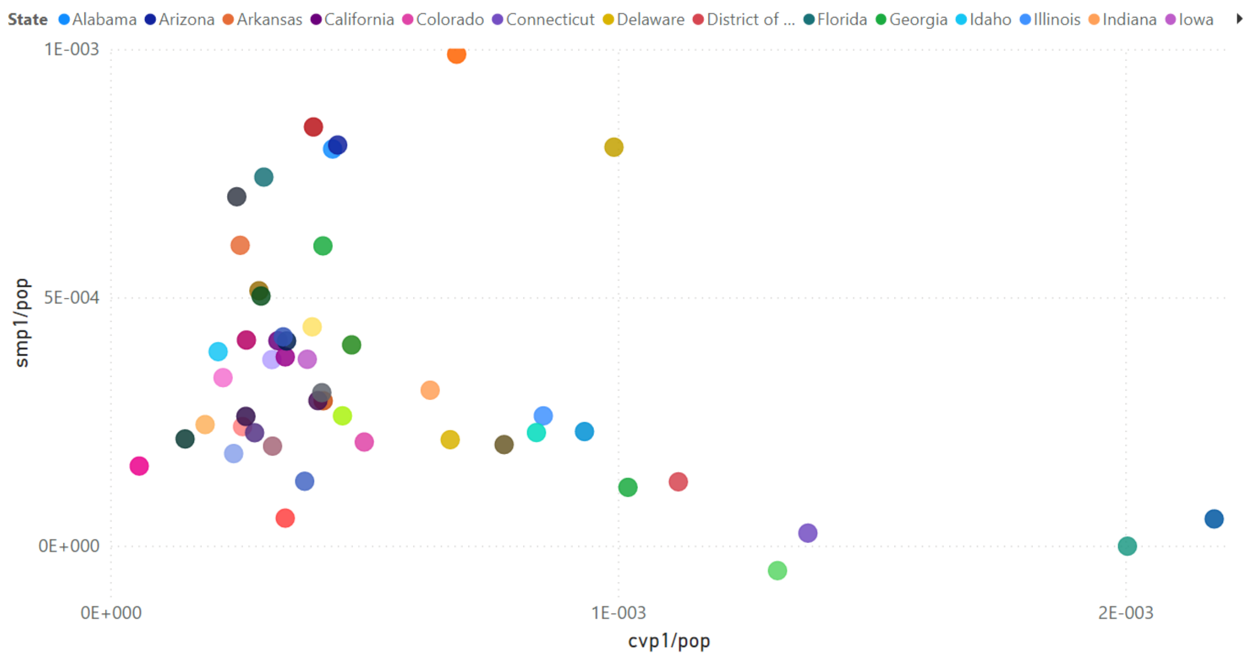


Figure 17a. smp1/pop versus cvp1/pop. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

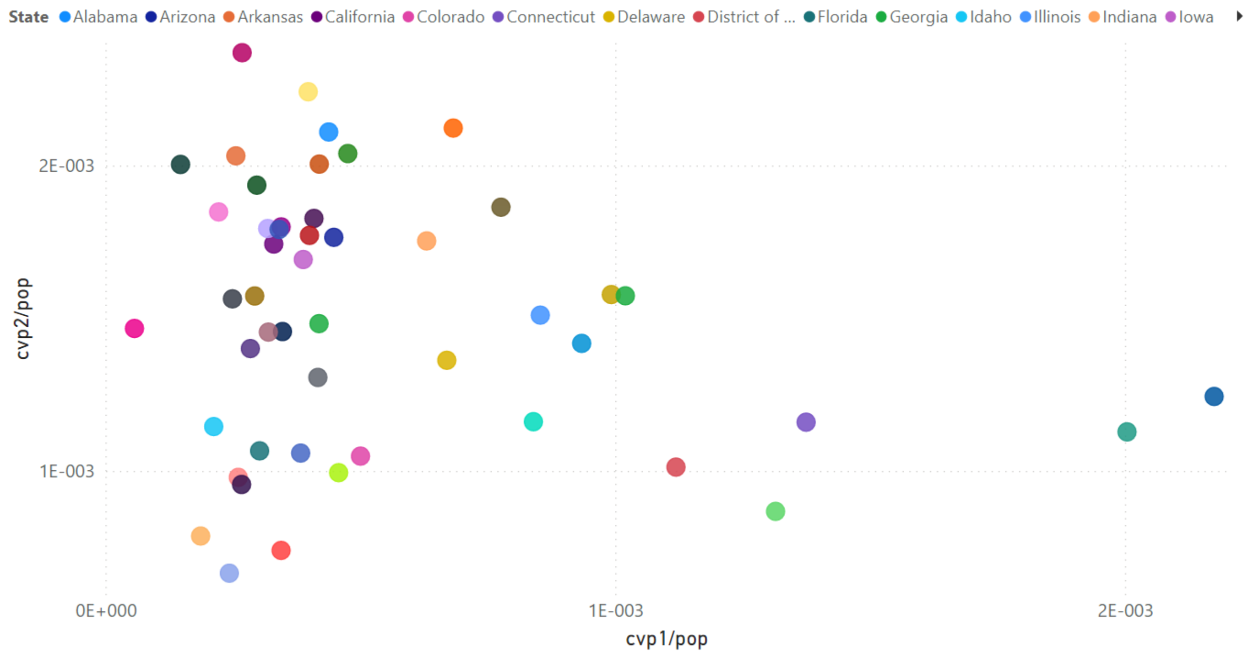


Figure 17b. cvp2/pop versus cvp1/pop. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

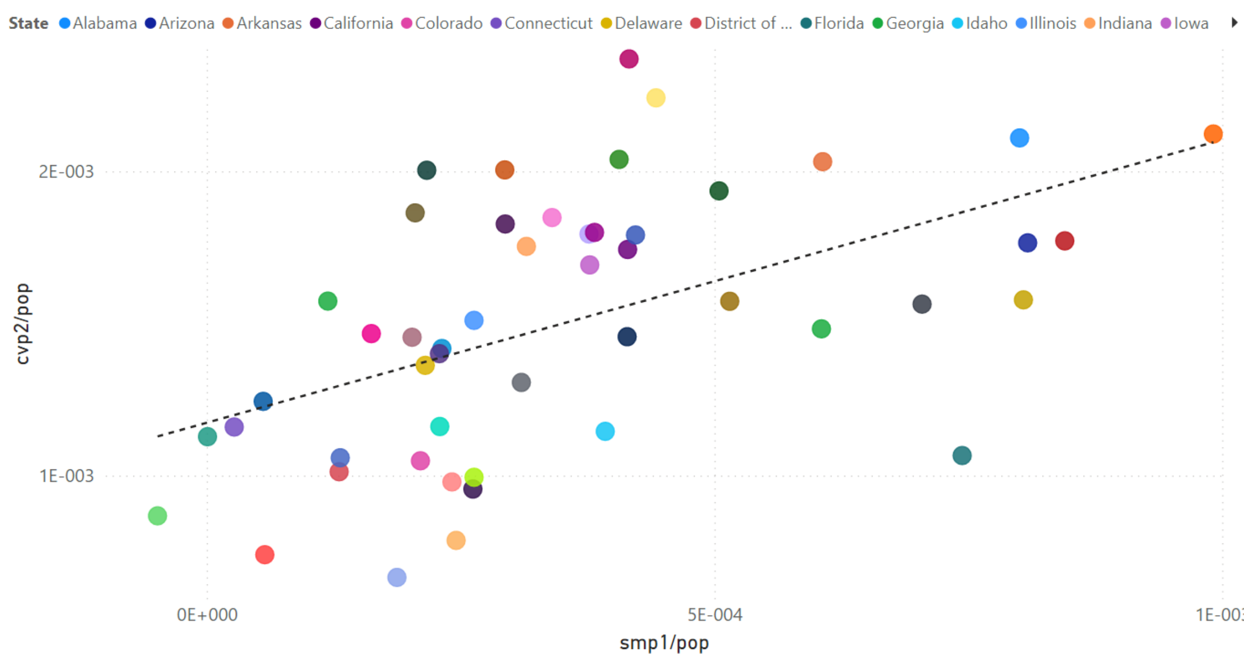


Figure 17c. cvp2/pop versus smp1/pop. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

Figure 17a shows that near-zero values of smp1/pop occur for the largest values of cvp1/pop, and that most large values of smp1/pop occur for small values of cvp1/pop. Similarly, Figure 17b shows that near-zero values of cvp2/pop occur for the largest values of cvp1/pop, and that most large values of cvp2/pop occur for small values of cvp1/pop.

This shows that the states with extremely large values of cvp1/pop (New York, New Jersey, Connecticut, Massachusetts… mainly the L0M pattern) had small (cvp2) or near-zero (smp1) values of mortality in the seasons that followed (summer-2020, fall-winter-2020-2021). Possible explanations include: the so-called "dry tinder" effect, in which those likely to die would have already died in the first "wave", or socio-geo-economic and climatic factors that give large smp1 and cvp2 are absent in those states that have the largest cvp1 peaks. Our analysis shows that the latter explanation is more likely. Indeed, different age groups, social classes (poverty, obesity) and state jurisdictions predominantly contribute to cvp1 versus smp1 and cvp2. A dry tinder effect interpretation for cvp1/smp1-cvp2 is not compatible with the many observed correlations.

A notable exception (outlier) in the smp1-cvp1 relation (Figure 17a) is Louisiana, which has both large cvp1 and large smp1. We have interpreted large values of cvp1 ("covid peak"), occurring heterogeneously and synchronously around the world, as being due to local-jurisdictional aggressive immediate medical and government responses to the 11 March 2020 WHO pronouncement of a pandemic (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021). New York City and New York state directives are the defining examples of such aggression. There is circumstantial evidence that Louisiana has a medico-government culture approaching that of New York: "Louisiana's largest hospital system will impose fee on employees if their spouse is unvaccinated", *Blaze media*, 01 October 2021, https://archive.ph/sDfL2.

Figure 17c shows that there is a correlation between cvp2/pop and smp1/pop. Such a correlation, as opposed to an anti-correlation, is contrary to a "dry tinder" effect

occurring between summer-2020 and fall-winter-2020-2021. Rather, it suggests that some or all of the same socio-geo-economic and climatic effects impact the mortality in both seasons.

The summer-2021 feature smp2 behaves similarly to smp1 (summer-2020) in many regards, although it starts later in the summer, and smp2/pop is correlated to smp1/pop, as shown in Figure 17d.

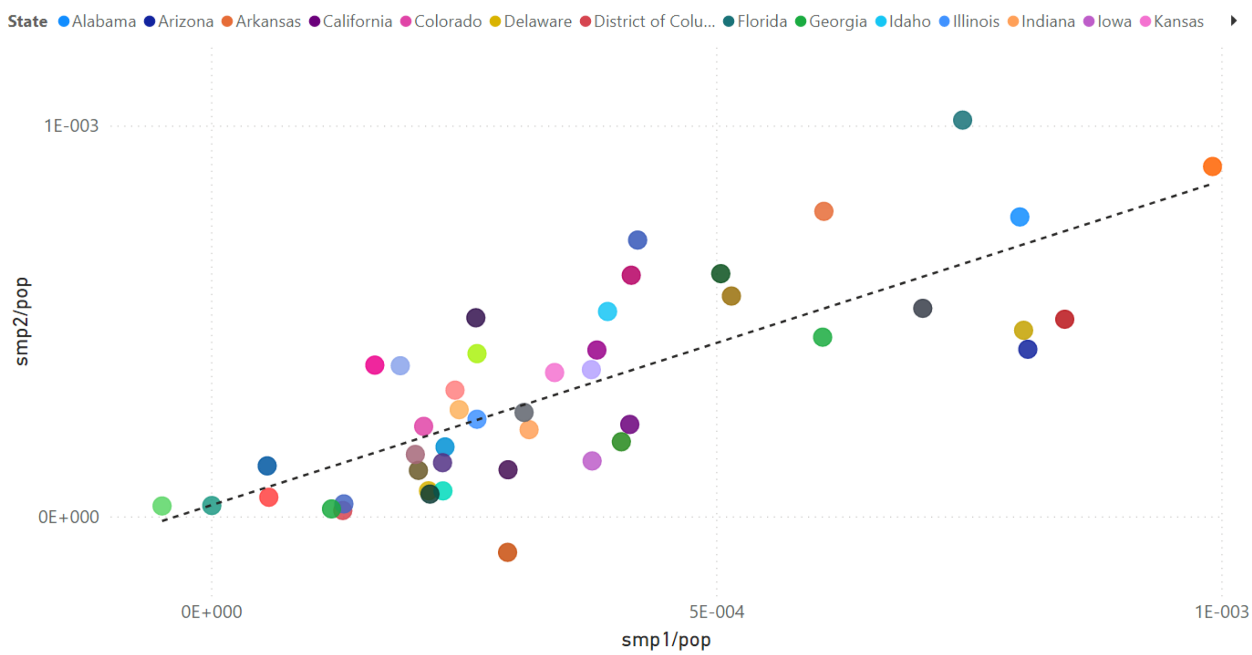


Figure 17d. smp2/pop versus smp1/pop. Each point is for one continental USA state. Connecticut, North Carolina and West Virginia are removed from the graph as there are not enough consolidated data points in ACM/w for smp2 for those states (see Appendix). The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

Figure 18 shows the same data as in Figure 17c, but with added circle-symbol-size (radius) determined by cvp1/pop.

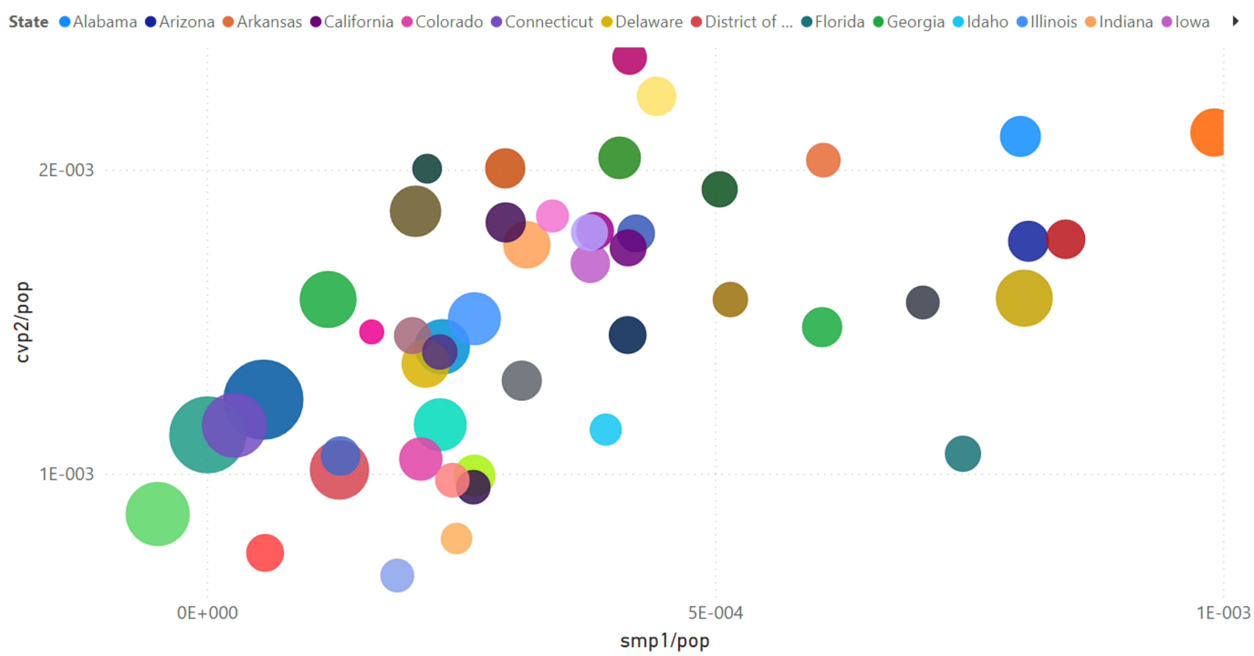


Figure 18. cvp2/pop versus smp1/pop, with the radius size determined by cvp1/pop. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

We note that the largest values of cvp1/pop (by state) are clustered at small values of both smp1/pop and cvp2/pop, with Louisiana as the main exception, followed by Mississippi.

3.8. Associations of COVID-era mortality outcomes with socio-geo-economic and climatic variables

The data, in which quantitative mortality outcomes (cvp1, smp1, cvp2, WB) are known by state, can be compared with state-wise or state-specific socio-geo-economic and climatic variables, in a search for correlations or relations, since all 49 diverse continental USA states can be used. This is a unique opportunity to identify factors which may cause or contribute to the excess (above-SB) USA mortality during the COVID-era.

We found three variables that appear to be determinative of COVID-era summer-2020 (smp1) and fall-winter-2020-2021 (cvp2) excess (above-SB) mortality in the USA. These are:

1. Climatic temperature (summer-period heatwave effect) (smp1)
2. Poverty (smp1 and cvp2)
3. Obesity (smp1 and cvp2)

The variables are somewhat correlated to each other, but have a significant degree of independence (one can be obese and rich, etc.). We found that using the product “OB.PV” of obesity (OB) and poverty (PV) gives a stronger correlation than either variable alone (being both obese and poor is deadlier than being either obese or poor).

We found that climatic temperature — evaluated using either maximum temperature (Tmax) or average temperature (Tav), either averaged in July-August-2020 or averaged over a calendar-year — is highly predictive of the geographical location of smp1 mortality (the hottest states were the most deadly in summer-2020, and dramatically so).

None of the variables (OB, PV, Tmax) that correlate with smp1 and cvp2 correlate with cvp1, which shows distinctly different death-causing phenomena in the two periods (cvp1 versus smp1-cvp2) in the COVID-era. We interpret cvp1 as being due to the immediate aggressive medical and government measures, whereas later deaths are apparently due to accumulated social and psychological chronic stress, combined with climatic stress, and affect younger individuals in broader age groups.

The latter age-dependence was shown by examining correlations between mortality outcomes and population age structure, by state. The smp1 feature (above-SB deaths in summer-2020) is uniquely anti-correlated with age of the state-wise population, which is contrary to WB mortality behaviour in all studied pre-COVID-era cycle-years, 2014-2019, and contrary to viral respiratory disease epidemiology.

Throughout this study, we compare our COVID-era results with a similar search for correlations in WB/pop mortality outcome in given cycle-years occurring prior to the COVID-era. Contrary to deaths in the COVID-era, normal epidemiology of the unperturbed society shows no state-to-state correlations of winter burdens with obesity, poverty or climatic temperature, whatsoever, in any of the six specific cycle-years 2014-2019. The only “normal era” correlation we find is with age structure, and it is persistent from year to year. The same is true for many more cycle-years for France, and so on. It seems clear to us that the variables obesity, poverty and climatic temperature become determinative, and have a disproportionate and immediate deadly impact, only in the significantly socio-economically perturbed and stressed population of the COVID-era measures.

Here are the details, as follows.

Obesity

Figure 19 shows the scatter plots for obesity (OB), defined as the prevalence of self-reported obesity among U.S. adults (CDC, 2021e).

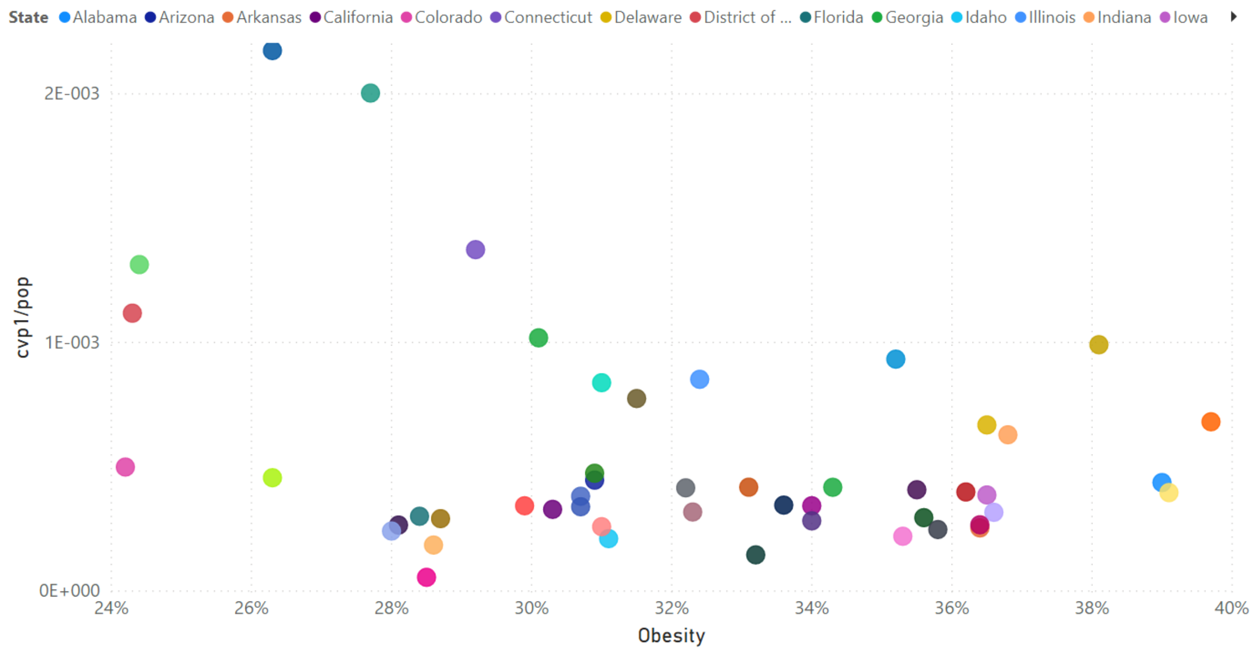


Figure 19a. cvp1/pop versus obesity. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is no discernable trend between cvp1/pop and OB.

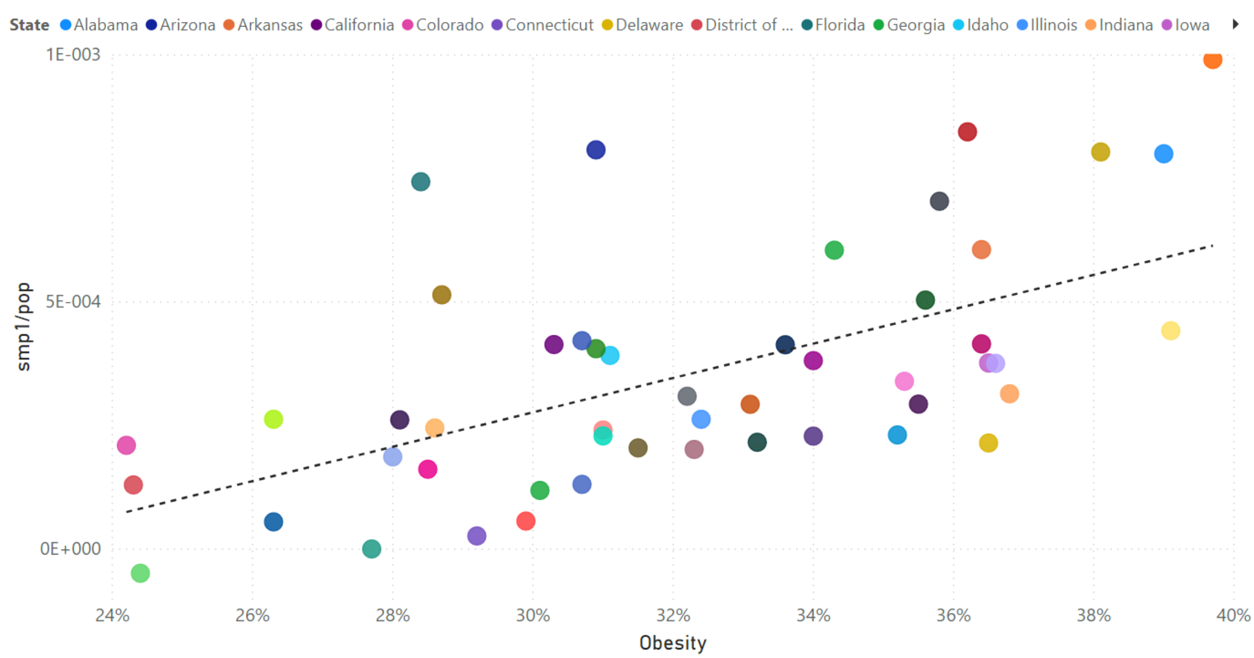


Figure 19b. smp1/pop versus obesity. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual

least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is a positive trend between smp1/pop and OB.

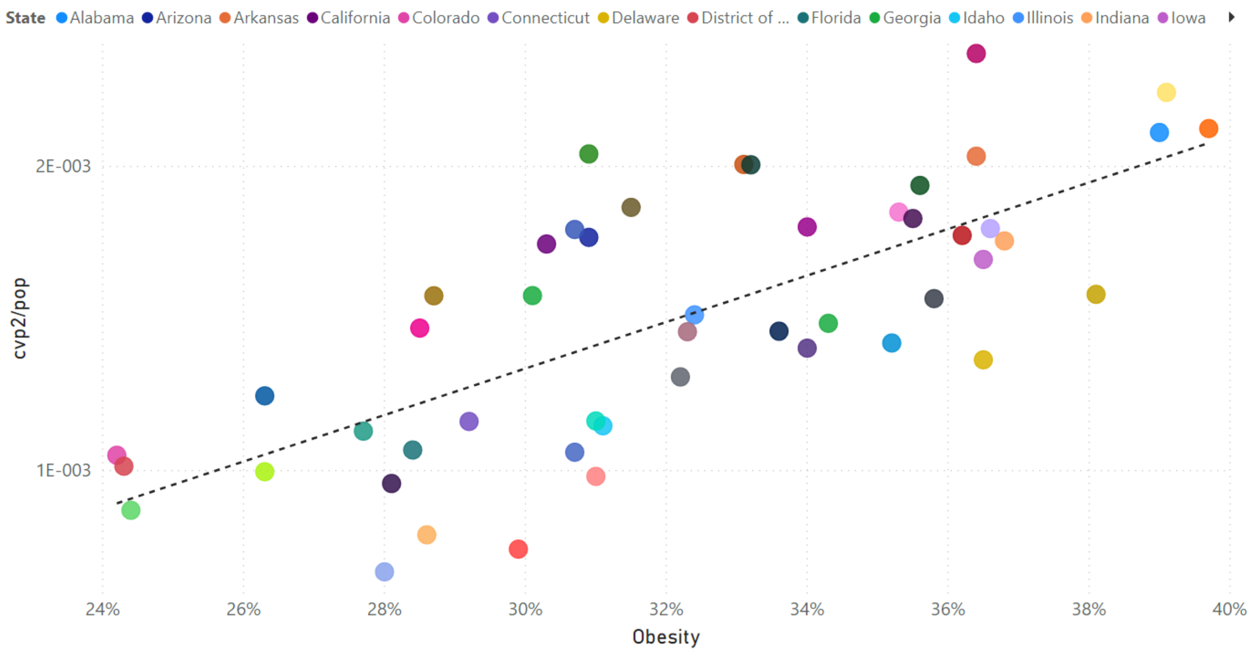


Figure 19c. cvp2/pop versus obesity. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is a positive trend between cvp2/pop and OB.

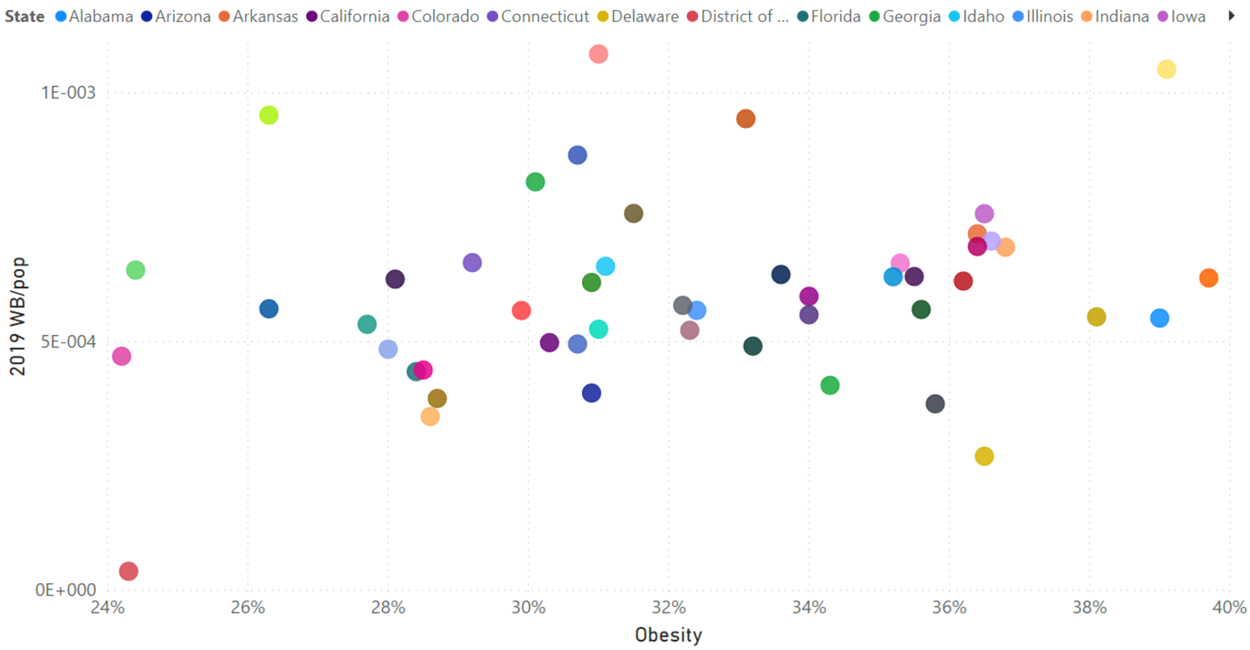


Figure 19d. WB/pop for cycle-year 2019 versus obesity. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is no correlation whatsoever. This is true for all pre-COVID-era cycle-years, 2014-2019 (data not shown). "Normal-era" winter burden deaths above-SB have no relation to obesity, on a state-wise basis.

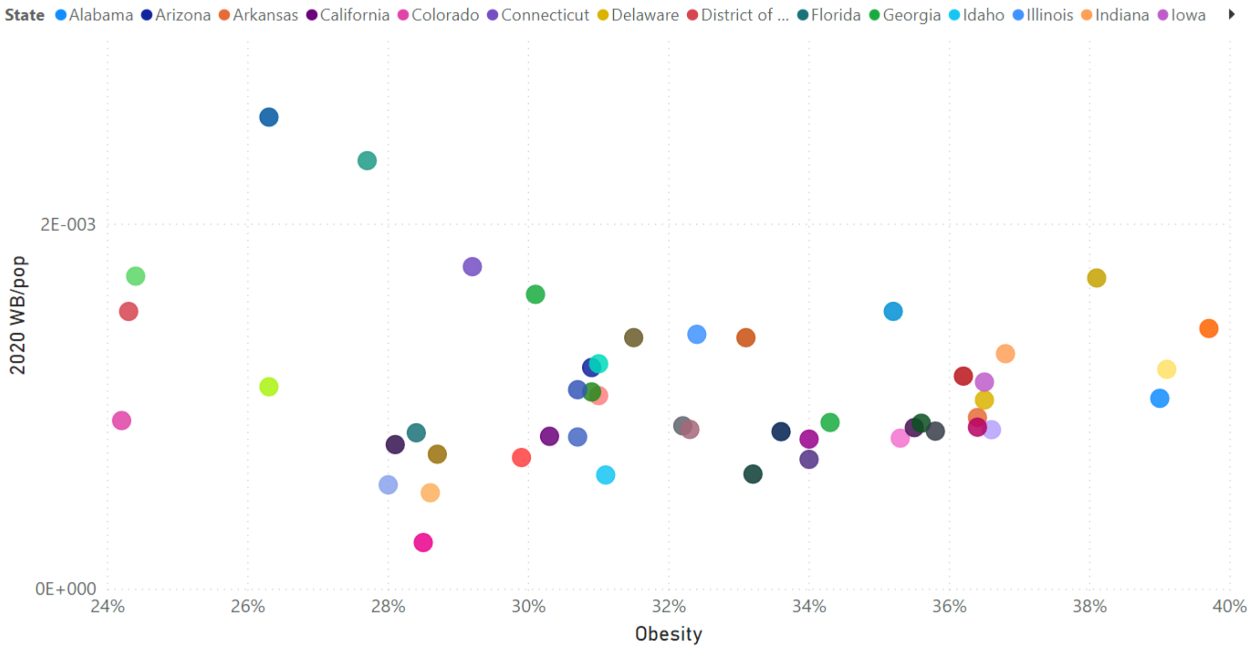


Figure 19e. WB/pop for COVID-era cycle-year 2020 versus obesity. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

Excluding the six states with highest 2020 WB/pop values and OB < 31 % (Connecticut, District of Columbia, Massachusetts, New Jersey, New York, Rhode Island), there is a positive trend for the remaining states. This is consistent with the fact that 2020 cycle-year includes both cvp1 and approximately half of smp1, and that the excluded states have extremely large cvp1/pop values in mostly wealthy states.

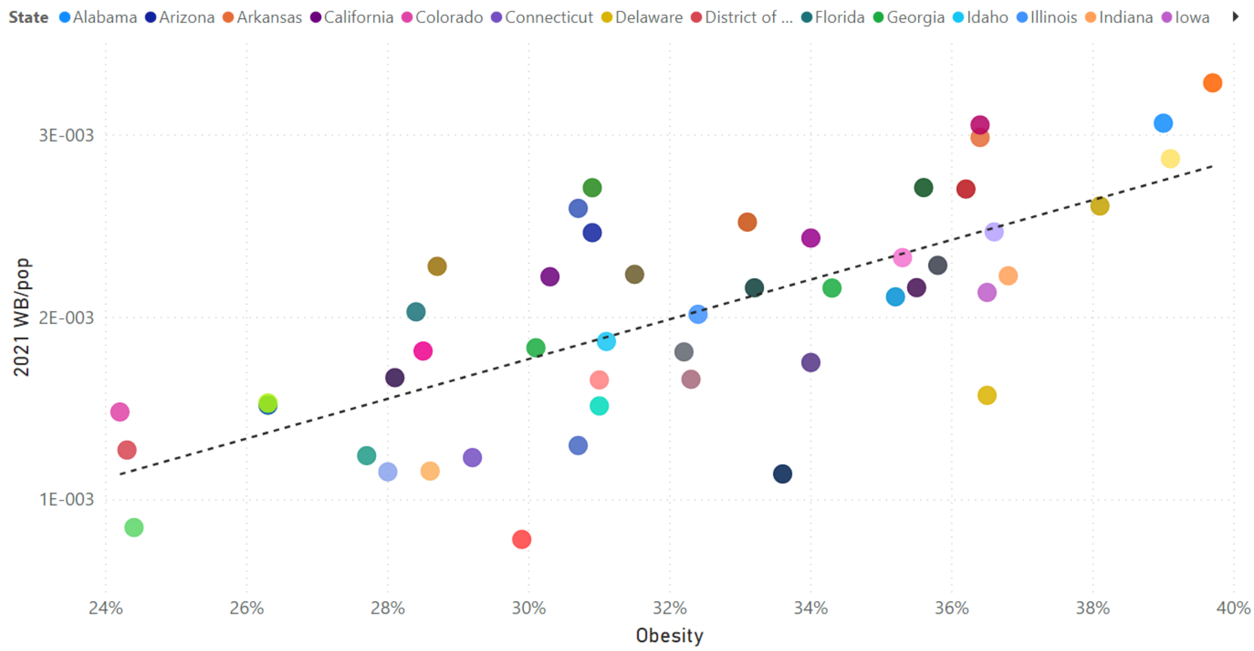


Figure 19f. WB/pop for COVID-era cycle-year 2021 versus obesity. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is a positive trend between WB/pop for COVID-era cycle-year 2021 and OB.

Poverty

Figure 20 shows the scatter plots for poverty (PV), defined as the estimated percent of people of all ages in poverty (US Census Bureau, 2021d).

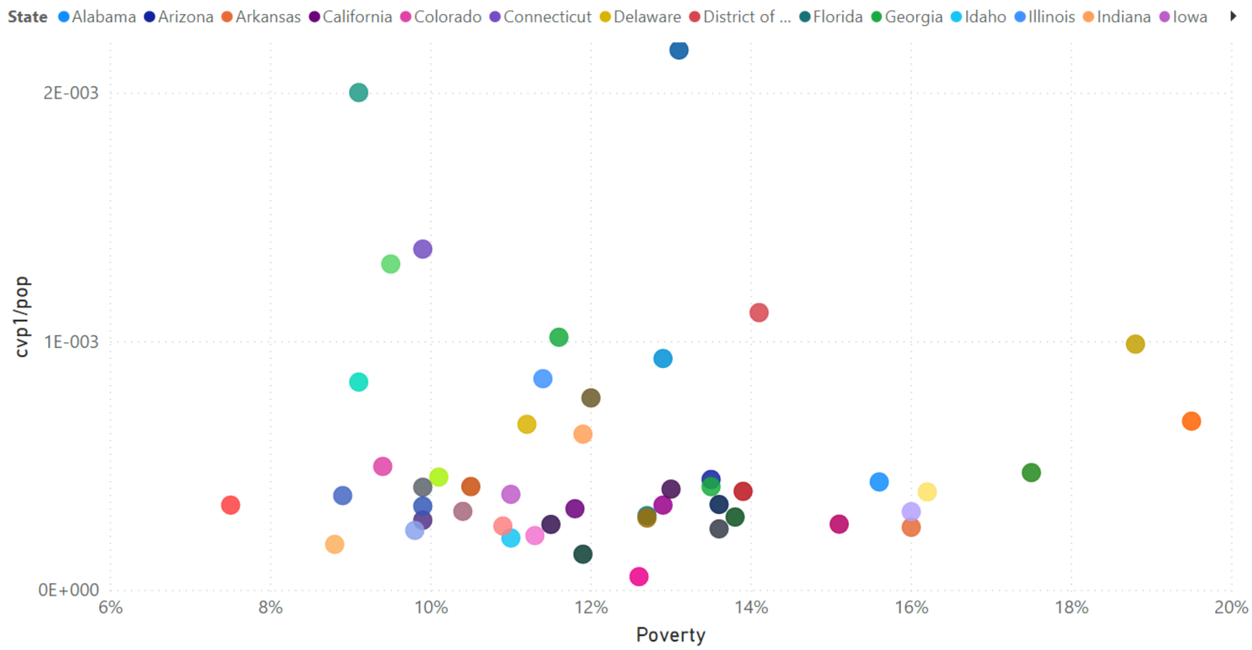


Figure 20a. cvp1/pop versus poverty. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is no discernable trend between cvp1/pop and PV.

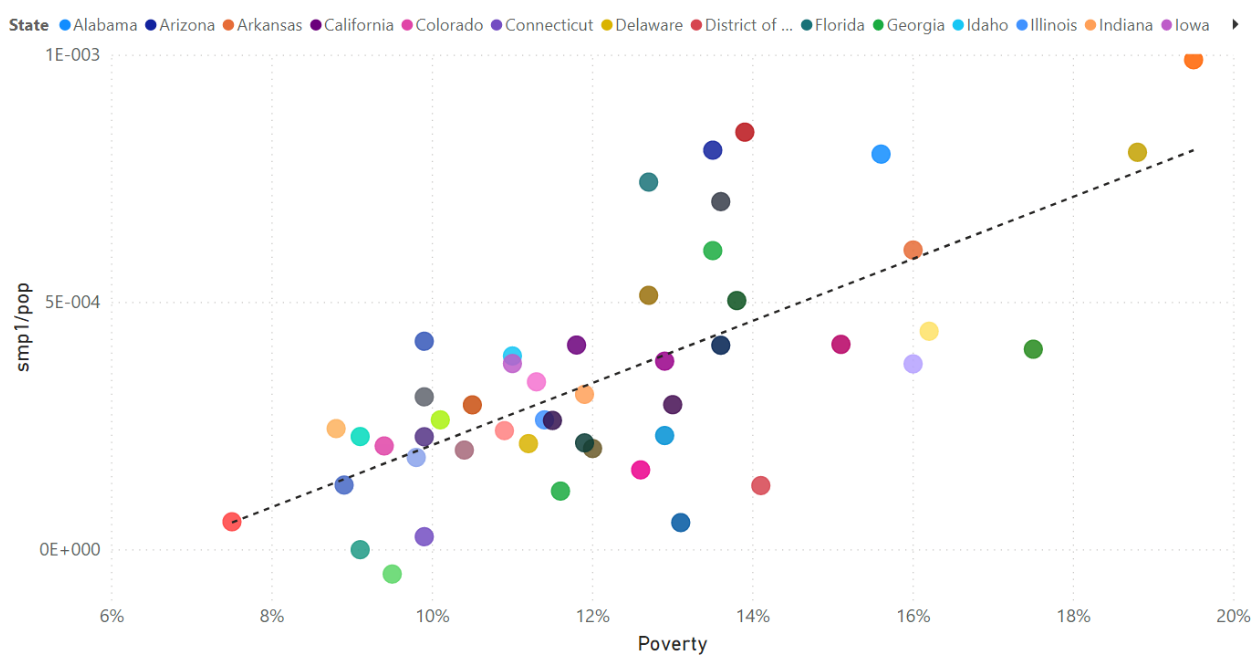


Figure 20b. smp1/pop versus poverty. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual

least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is a positive trend between smp1/pop and PV.

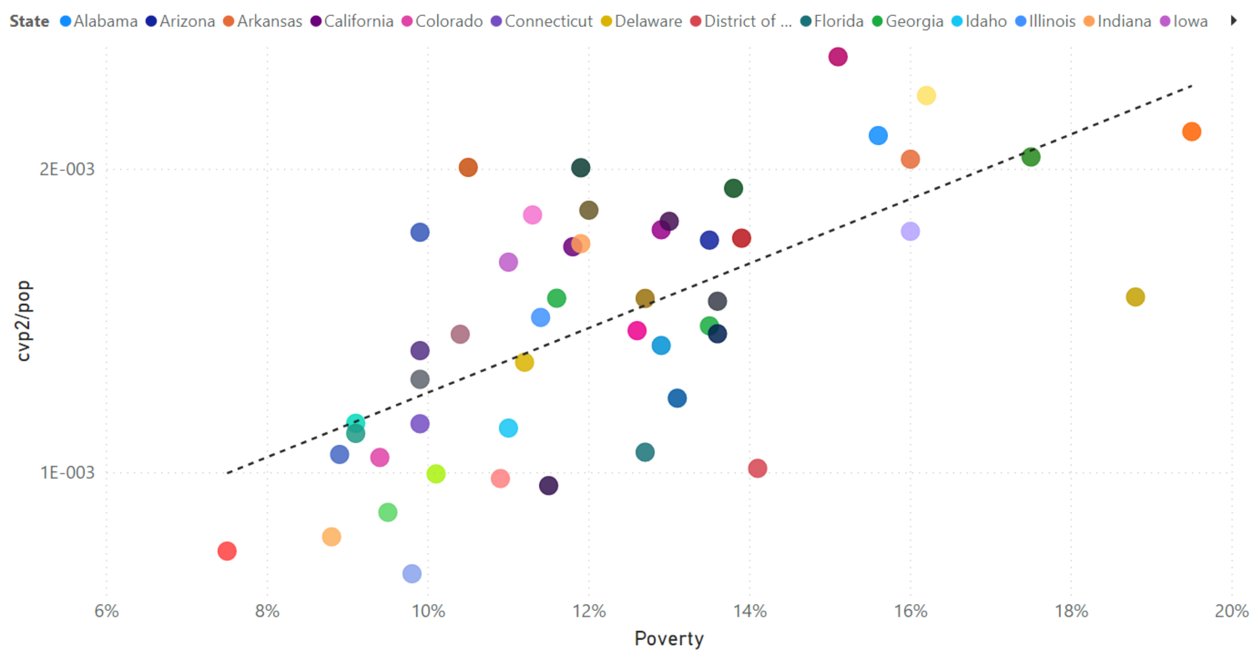


Figure 20c. cvp2/pop versus poverty. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is a positive trend between cvp2/pop and PV.



Figure 20d. WB/pop for cycle-year 2019 versus poverty. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is no correlation whatsoever. This is true for all pre-COVID-era cycle-years, 2014-2019 (data not shown). "Normal-era" winter burden deaths above-SB have no relation to poverty, on a state-wise basis.

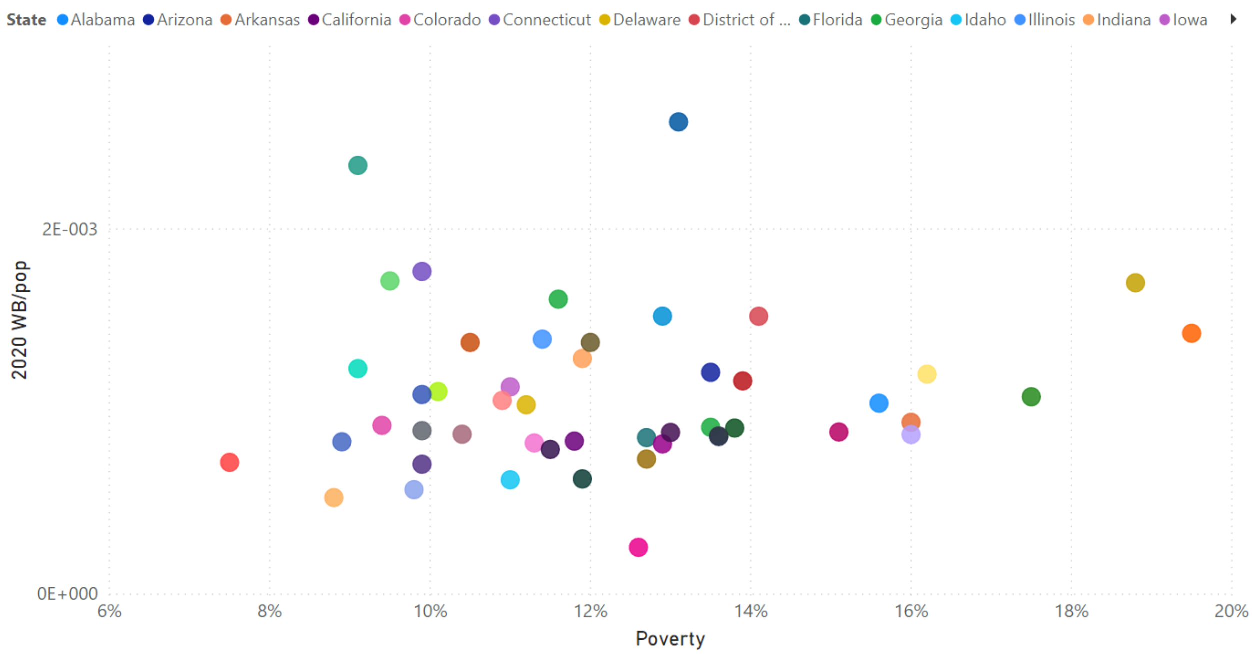


Figure 20e. WB/pop for COVID-era cycle-year 2020 versus poverty. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

Excluding the four states with highest 2020 WB/pop values (Connecticut, Massachusetts, New Jersey, New York), there is a positive trend for the remaining states. This is consistent with the fact that 2020 cycle-year includes both cvp1 and approximately half of smp1, and that the excluded states have extremely large cvp1/pop values in mostly wealthy states.

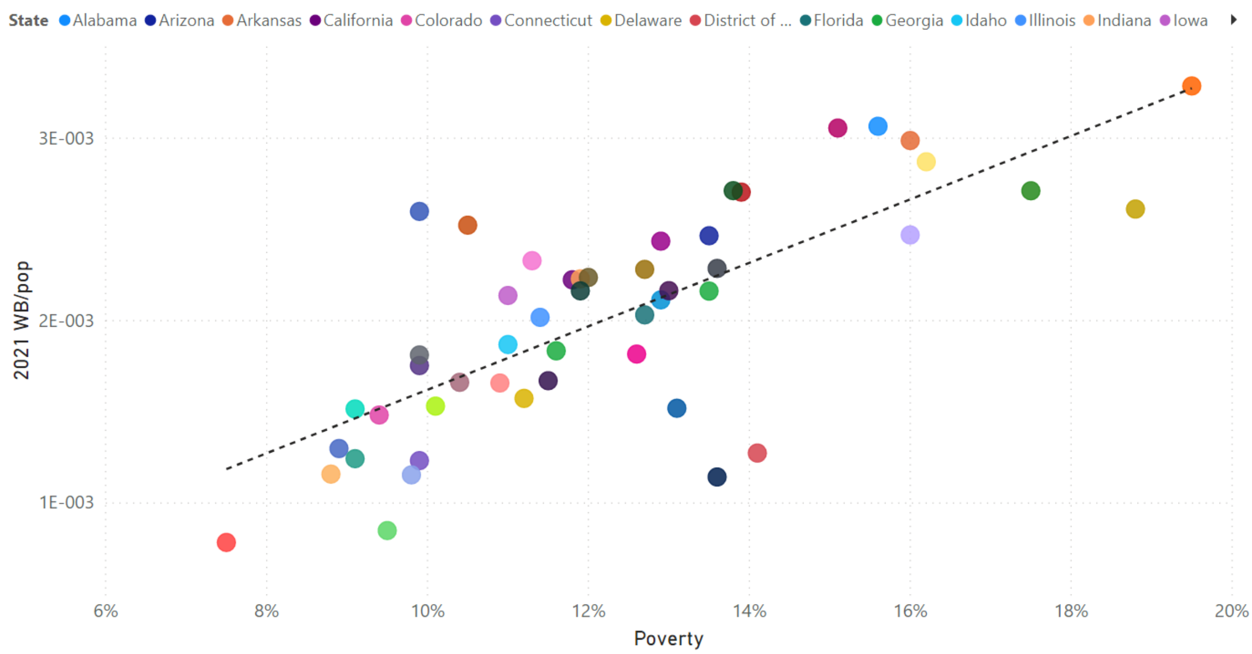


Figure 20f. WB/pop for COVID-era cycle-year 2021 versus poverty. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is a positive trend between WB/pop for COVID-era cycle-year 2021 and PV. The outlier at 13.6 % poverty is North Carolina, which is an artifact of incomplete data for the final weeks for this state (see Appendix).

Climatic temperature

One of the most striking results of our study is that the summer-2020 excess (above-SB) mortality is concentrated in Southern states (Figure 16). Excess summer mortality is striking in itself because viral respiratory diseases barely transmit in humid summer climates (aerosol particles are not stable in high absolute humidity: Harper, 1961; Shaman et al., 2010), and summers “always” exhibit seasonal lows of mortality in mid-latitude regions, seasonally inverted in the Southern hemisphere. Yet, here in the USA, there was an actual peaked maximum in ACM/w in the summer-2020 (Figures 5, 6, 7, 9, 10, and Appendix).

The geographical pattern of summer-2020 excess (above-SB) mortality, on a map of the USA (Figure 16), is remarkably well predicted by climatic temperature, shown in Figure 21.

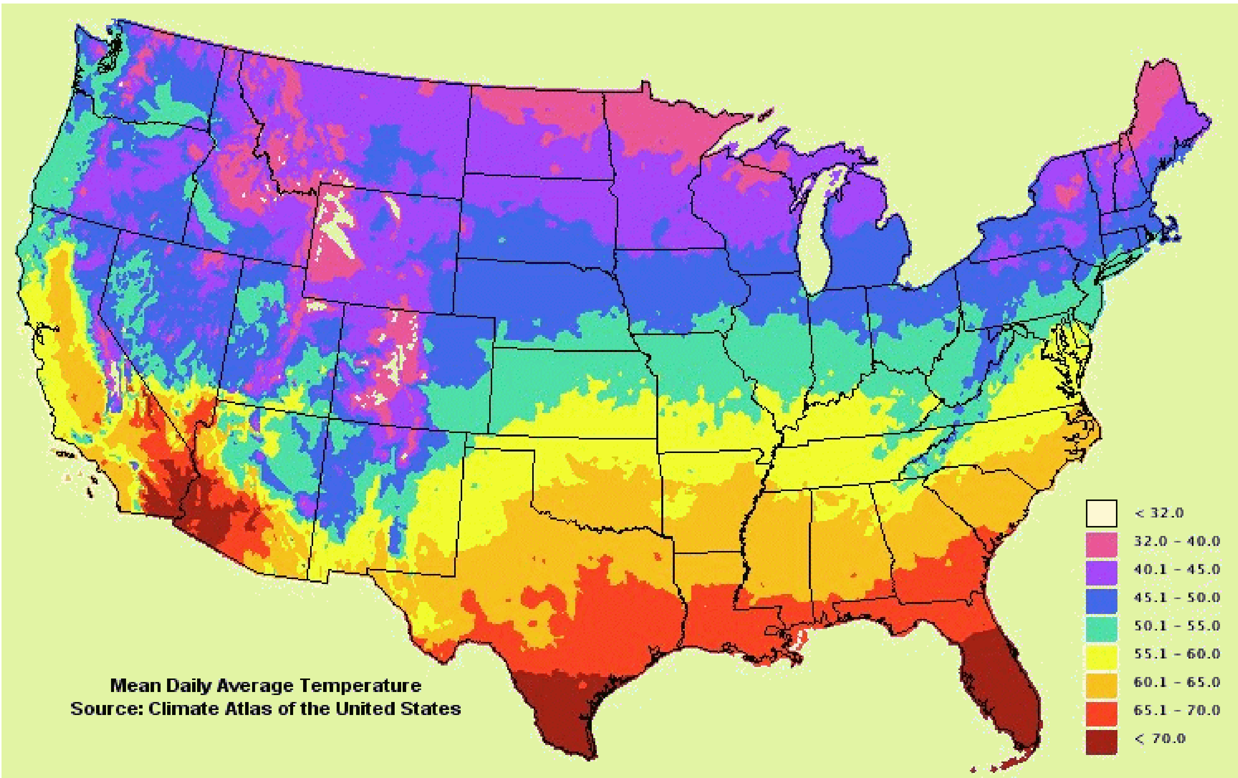


Figure 21. Mean daily average temperature: Mean of daily minimum and maximum, averaged over the year, and for three decades (1970-2000). This represents "climatic mean temperature" for the continental USA (spatial average is achieved using weighted cells, with the available surface air weather stations). Source: Climate Atlas of the United States, developed by NOAA's National Climatic Data Center in Asheville, NC., Version 2.0, CD-ROM, released September 2002. Figure accessed at http://www.virginiaplaces.org/climate/ on 26 September 2021. (Typo: "< 70.0" should be "> 70.0").

We illustrate this on a state-by-state basis, using the state-wise average August-2020 temperature, shown in Figure 22.

AVERAGE TEMPERATURE, AUGUST 2020

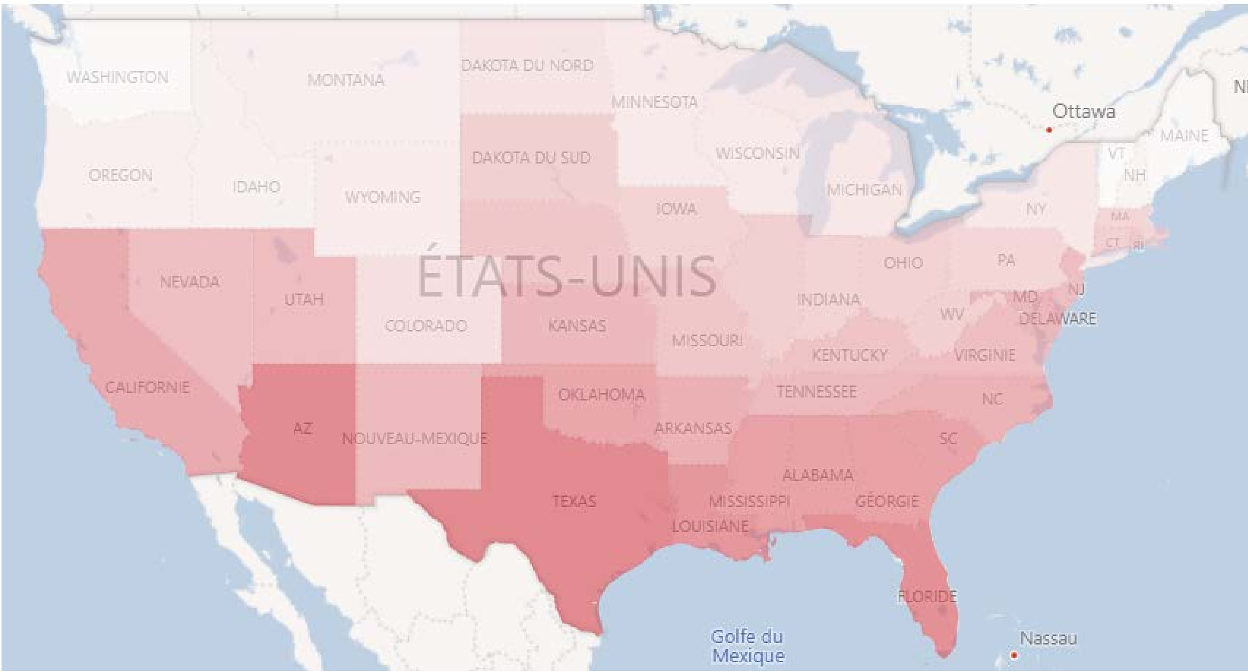


Figure 22. Average temperature, per state of the continental USA, for August 2020. Continental USA excludes Alaska and Hawaii. The darker the red, the higher the average temperature. Climatic temperature data were retrieved from the NOAA (NOAA, 2021), as described in Table 1. (The reader is asked to compare this map with the map shown in Figure 16.)

Essentially the same pattern occurs for July 2020, or for any month, or for yearly averages, or using daily maximum temperatures rather than daily average temperatures. Basically, all the average temperatures (averages of daily averages, or averages of daily maxima; on July or August, or on July and August, or on any calendar-year or cycle-year) chosen to represent climatic temperature are highly correlated to each other. For our purpose, these different averages are interchangeable.

The correlation between climatic temperature and summer-2020 excess (above-SB) mortality (smp1/pop, by state) is illustrated in Figure 23, using the July-August 2020 average daily maximum temperature (averaged by state and over the two-month period).

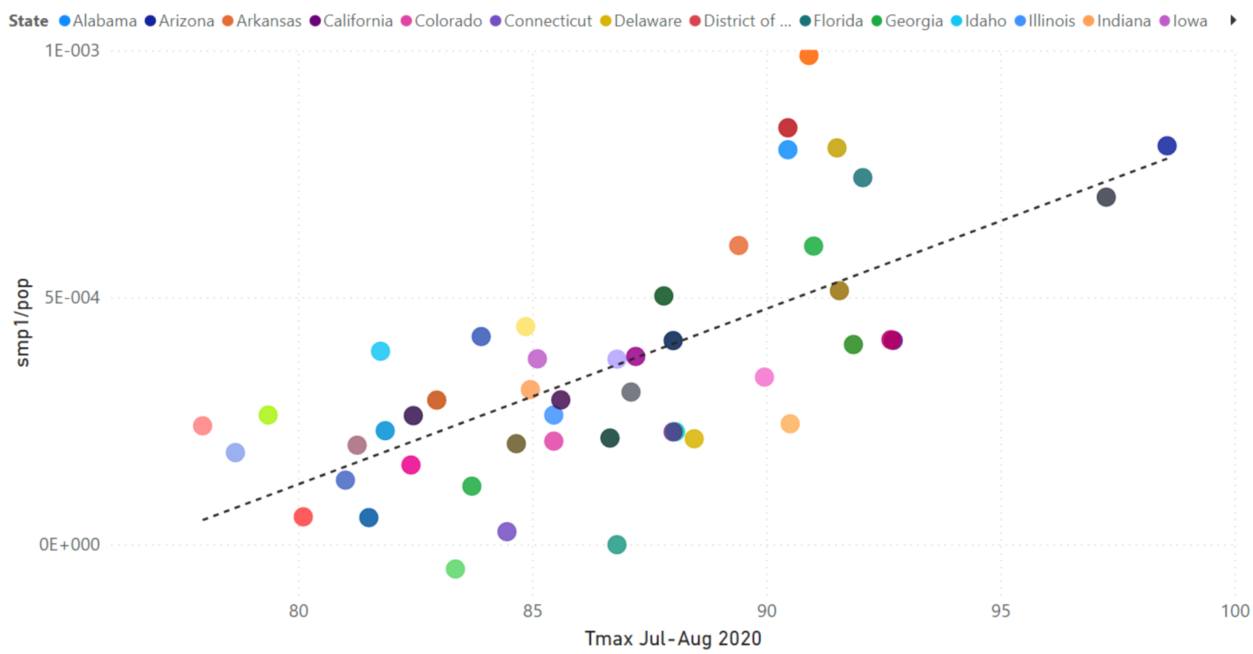


Figure 23. smp1/pop versus average daily maximum temperature over July and August 2020, Tmax Jul-Aug 2020. Each point is for one continental USA state, excluding District of Columbia, for which no temperature data were available (NOAA, 2021). The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

There is a clear positive trend. Here (Figure 23), the four main high-smp1/pop-value outliers are Mississippi, South Carolina, Alabama and Louisiana; whereas the three main low-smp1/pop-value outliers are Massachusetts, Connecticut and New Jersey.

Such a trend between an excess (above-SB) mortality and mean temperature, per state, does not exist, whatsoever, in the winter burden mortality (WB/pop) for any of the pre-COVID-era cycle-years, 2014-2019 (data not shown).

Obesity, poverty, and climatic temperature

Next, we examine the above correlations further. Figure 24 shows that obesity (OB) and poverty (PV) are somewhat correlated to each other.

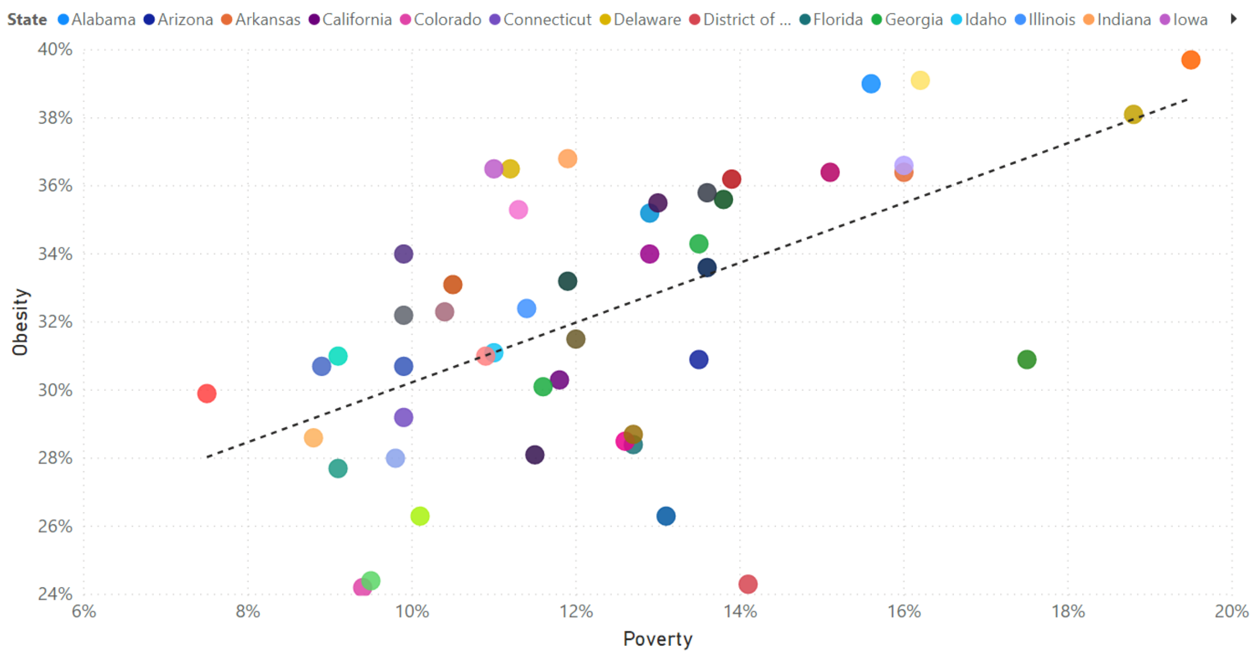


Figure 24. Obesity versus poverty. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved as described in section 2.

Given the above, we decided to try using the product of obesity and poverty (OB.PV) as a variable. Figure 25 shows smp1/pop versus OB.PV, with added circle-symbol-size (radius) determined by the July-August 2020 average daily maximum temperature (averaged by state and over the two-month period).

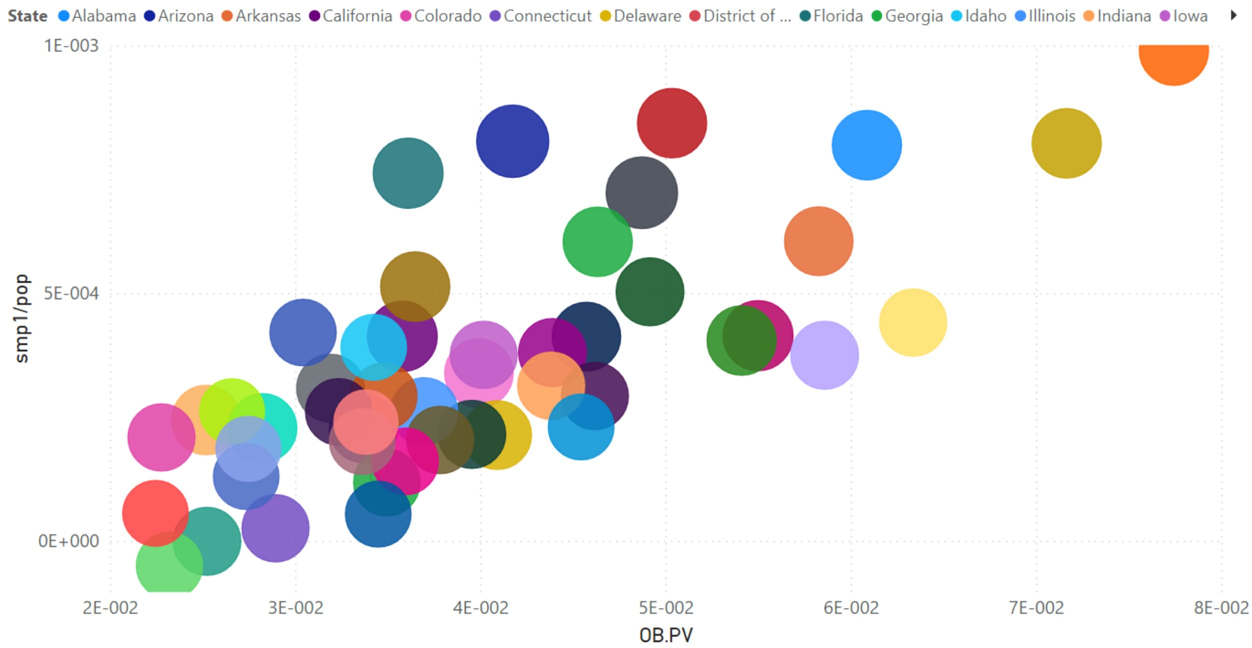


Figure 25. smp1/pop versus the product of obesity and poverty (OB.PV), with the radius size determined by Tmax Jul-Aug 2020. Each point is for one continental USA state, excluding District of Columbia, for which no temperature data were available (NOAA, 2021). The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

The correlation is excellent. Climatic temperature (circle size) also appears to be correlated to OB.PV (Figure 25). Figure 26 shows the average of daily average temperatures over the calendar-year 2020 (Tav 2020) versus OB.PV, with added circle-symbol-size (radius) determined by the outcome smp1/pop.

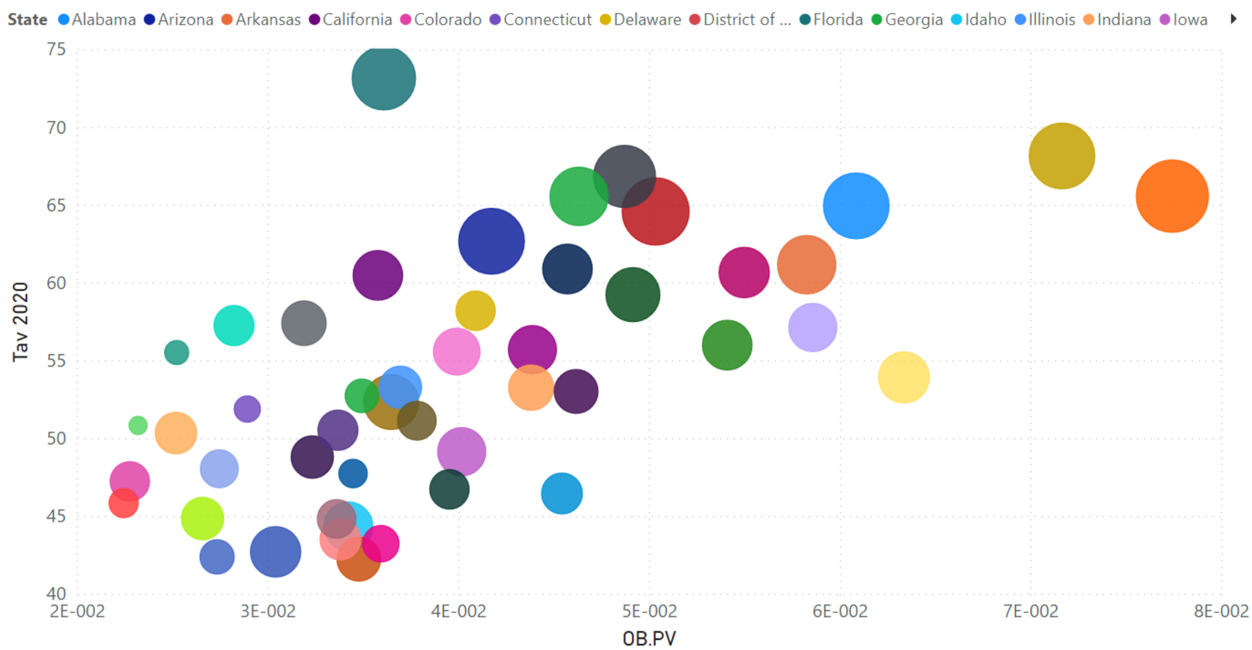


Figure 26. Tav 2020 versus the product of obesity and poverty (OB.PV), with the radius size determined by smp1/pop. Each point is for one continental USA state, excluding District of Columbia, for which no temperature data were available (NOAA, 2021). The colour-code of the other 48 continental states is shown in section 2. Data were retrieved as described in section 2.

Figure 26 shows two things.

First, climatic temperature is correlated to the product OB.PV.

Second, a diagram of climatic temperature versus OB.PV provides a strong predictor of whether there will be large summer mortality following an extended period of chronic psychological stress applied to the population.

Age structure of the population

More than 60 % of COVID-assigned deaths in the USA occur in the 85+ years age group (Kostoff et al., 2021; their Figure 1). The same is generally true of all viral respiratory diseases in Western nations.

Figure 27 shows WB/pop versus percent of population consisting of 85+ year olds ("85+/pop"), for each pre-COVID-era cycle-year, 2014-2019. The latter percentage more than doubles across all states, from approximately 1.2 % to approximately 2.6 %. Whereas the illustrated correlation is weak, it is persistently positive, having similar slope magnitudes, across all cycle-years, except for cycle-year 2016 (Figure 27c) where the nominally positive correlation (not shown) is not statistically meaningful.

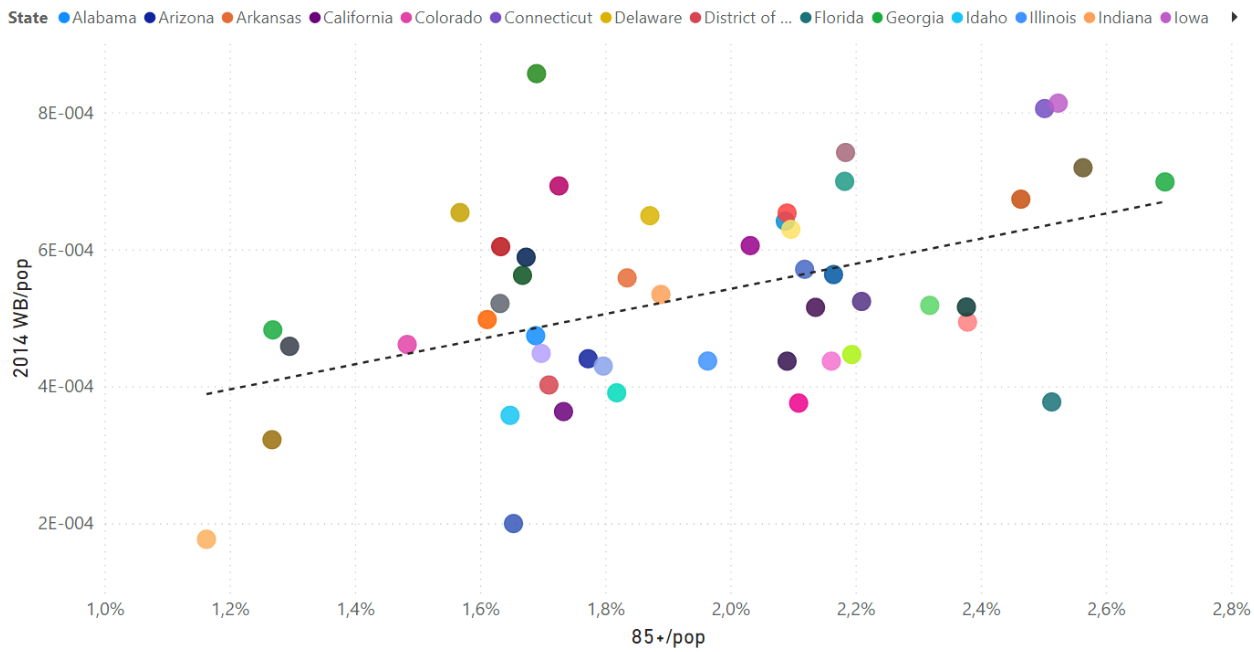


Figure 27a. WB/pop versus 85+/pop for cycle-year 2014. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2. Outliers: Utah (bad data point in 2014), Wyoming (less populous state, poor statistics, underestimation of SB).

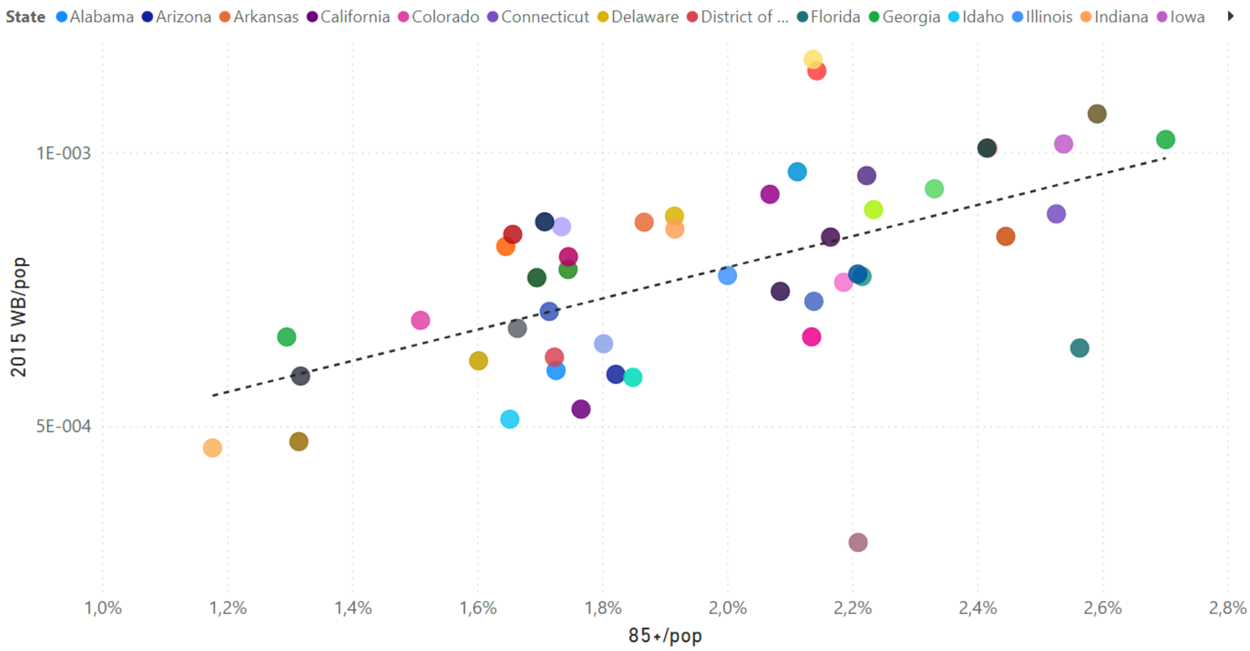


Figure 27b. WB/pop versus 85+/pop for cycle-year 2015. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2. The outlier Wisconsin is due to bad data points in 2015 for this state (see Appendix).

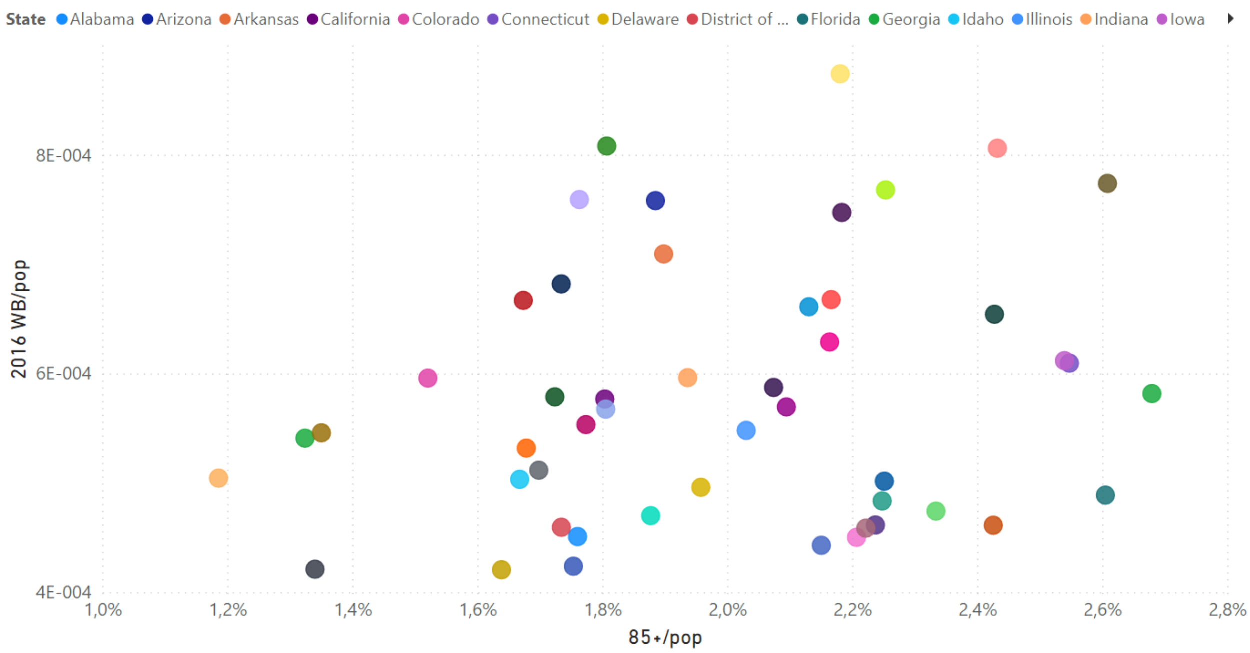


Figure 27c. WB/pop versus 85+/pop for cycle-year 2016. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

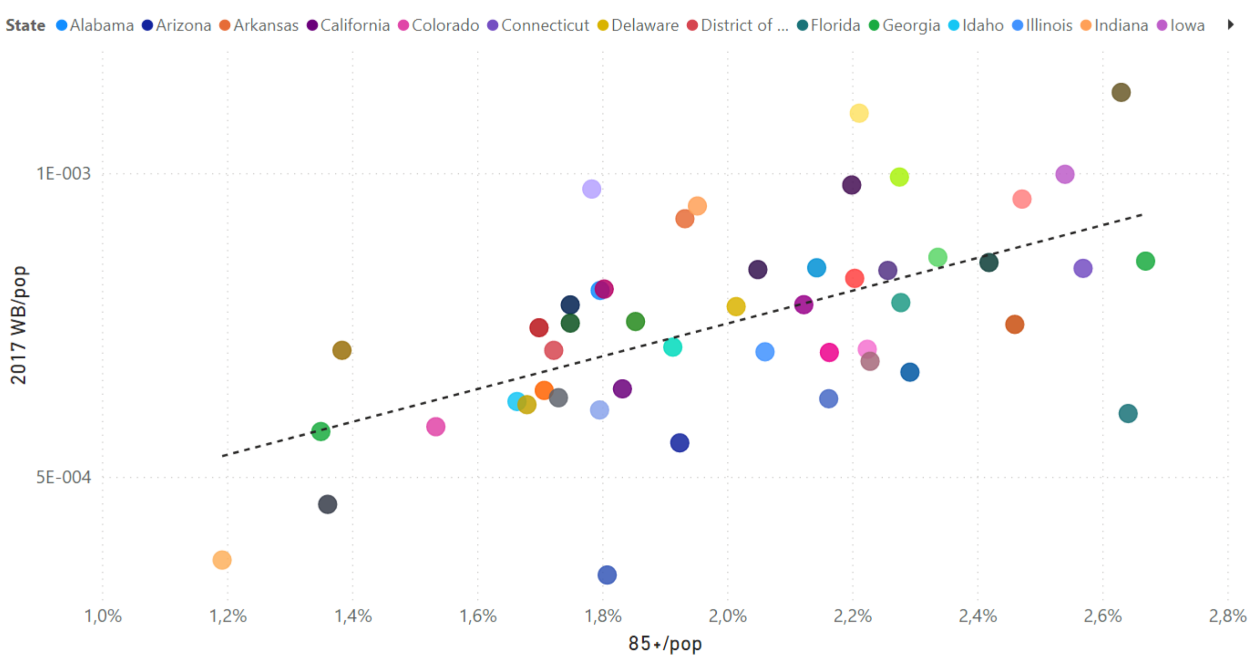


Figure 27d. WB/pop versus 85+/pop for cycle-year 2017. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2. Outlier: Wyoming (less populous state, poor statistics).

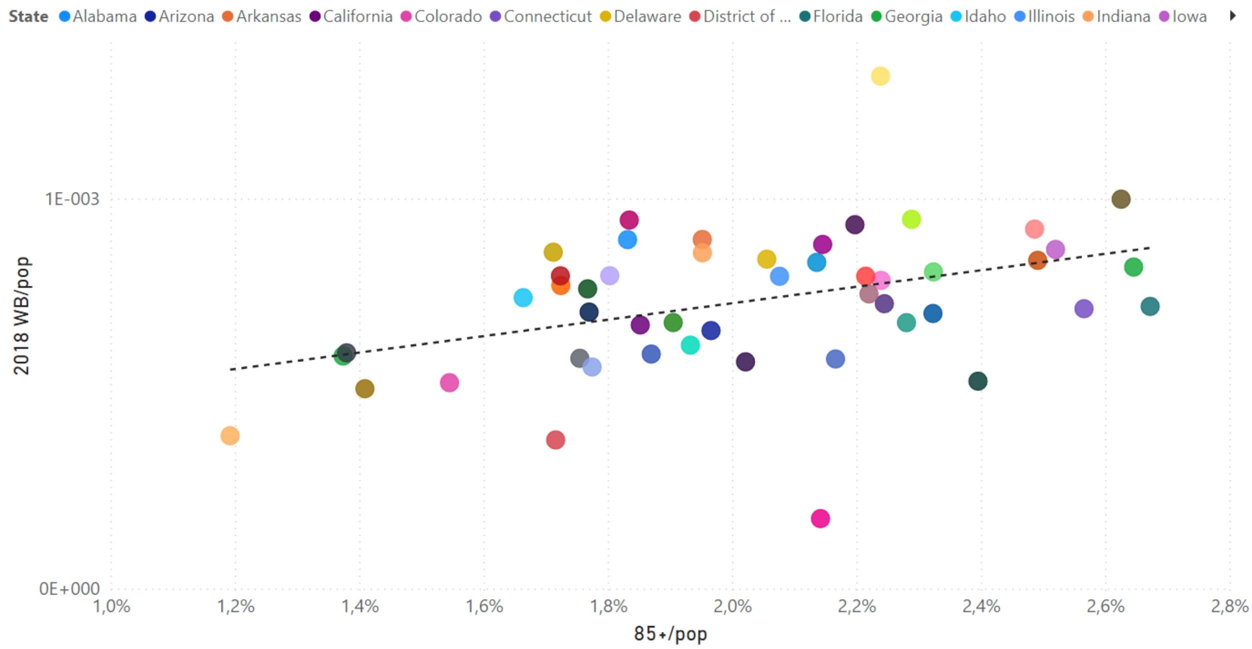


Figure 27e. WB/pop versus 85+/pop for cycle-year 2018. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2. Outliers: West Virginia (underestimation of SB, overestimation of WB), Montana (reverse).

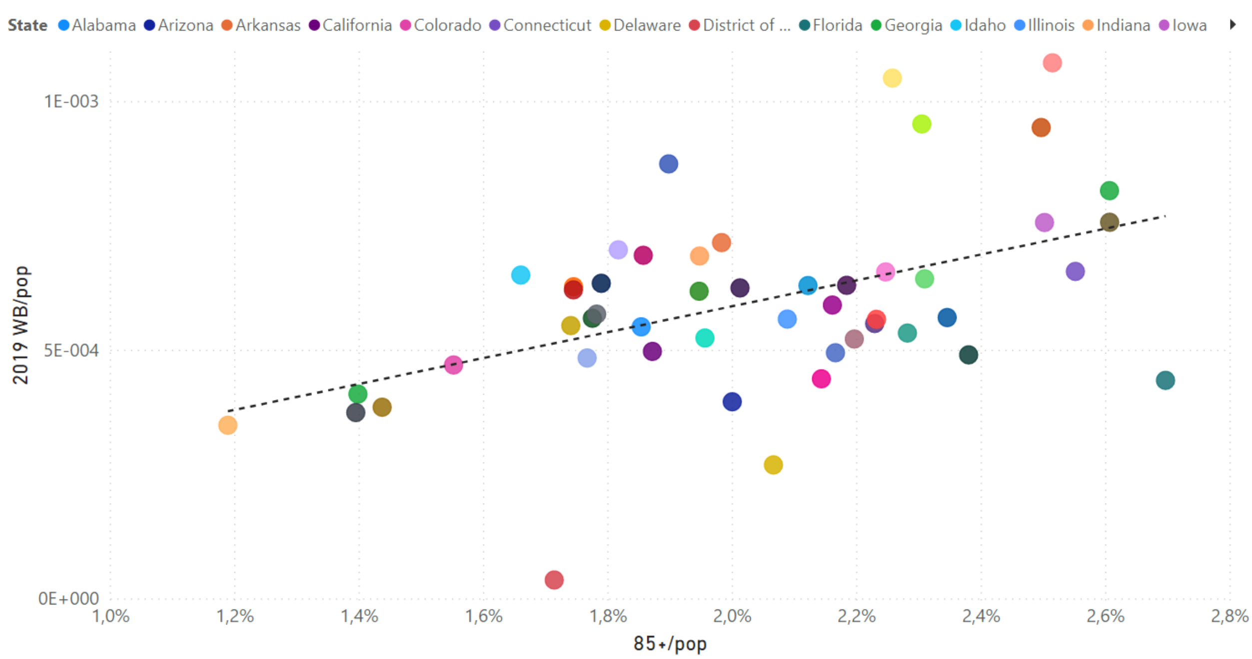


Figure 27f. WB/pop versus 85+/pop for cycle-year 2019. Each point is for one continental USA state. Outlier: District of Columbia (small state, poor statistics).

The same phenomenon (positive correlation of WB/pop with population fraction of the age group, in the pre-COVID-era cycle-years) occurs for all the older age groups: 45-54, 55-64, 65-74, 75-84, and 85+ ages. The correlation is then negative (anti-correlation) for 35-44 years, and not discernable for younger age groups (data not shown).

This age-dependence of winter burden mortality was expected, and is well known. Young people do not generally die of viral respiratory diseases that are prevalent in the winter.

In the COVID-era, cvp1/pop does not have a statistically meaningful correlation with 85+/pop, as shown in Figure 28a. It might best be described as no correlation whatsoever for states having essentially zero-magnitude cvp1/pop values, and several randomly placed outliers above the group having near-zero values of cvp1/pop. This is consistent with the idea that the cvp1 feature is predominantly due to the jurisdiction-specific response to the declaration of a pandemic.

Surprisingly, however, the summer-2020 excess (above-SB) mortality (smp1/pop) has an anti-correlation ("neg-cor") with 85+/pop, again with significant outliers, as shown in Figure 28b; and the fall-winter-2020-2021 mortality (cvp2/pop) has no discernable correlation with 85+/pop, as shown in Figure 28c. Correspondingly, the WB/pop versus 85+/pop has a positive correlation for cycle-year 2020 (Figure 28d), and a uniquely strong negative (anti-)correlation for cycle-year 2021 (Figure 28e).

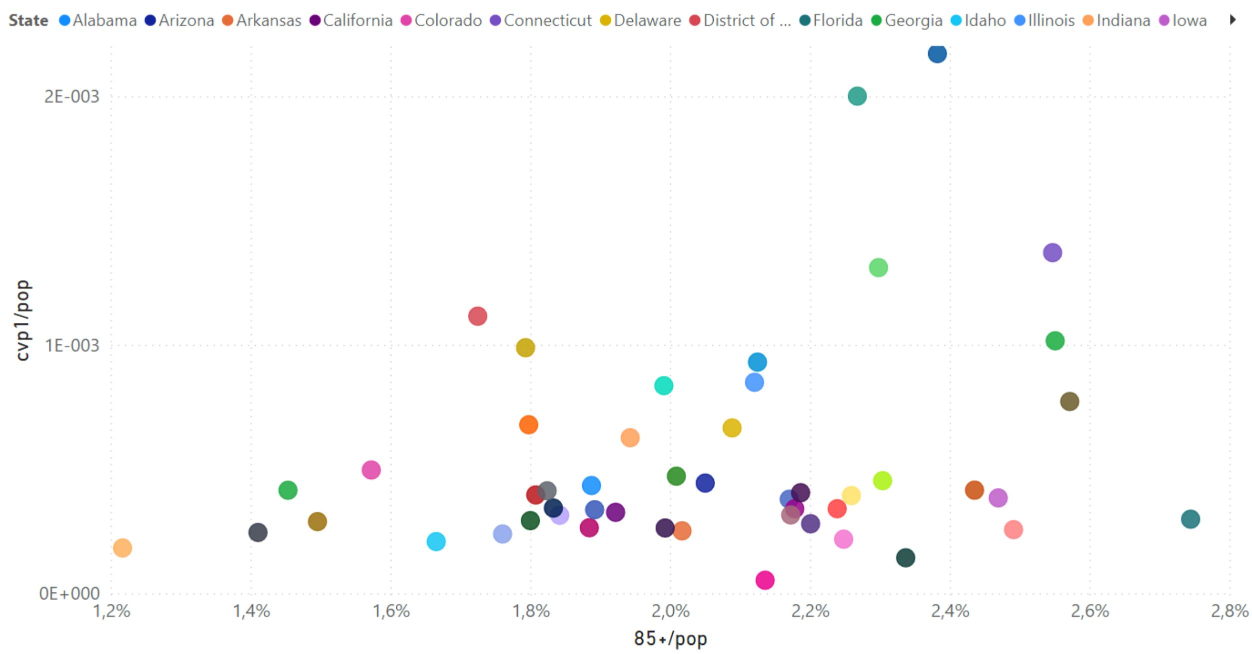


Figure 28a. cvp1/pop versus 85+/pop. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

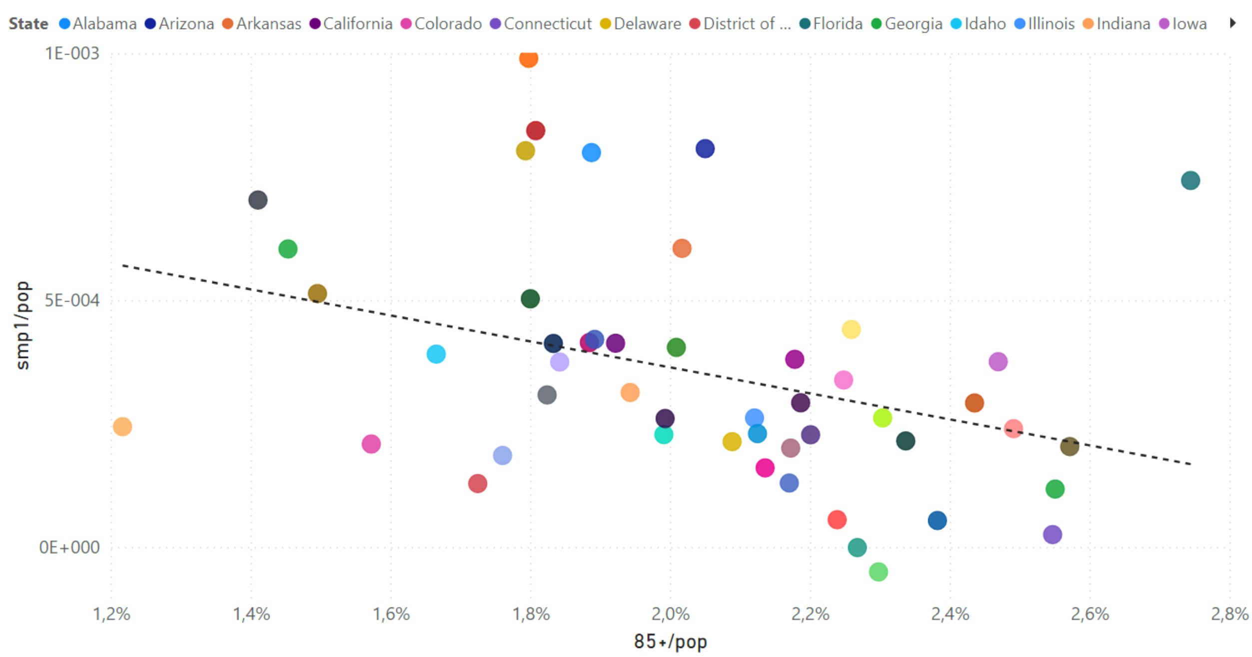


Figure 28b. smp1/pop versus 85+/pop. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

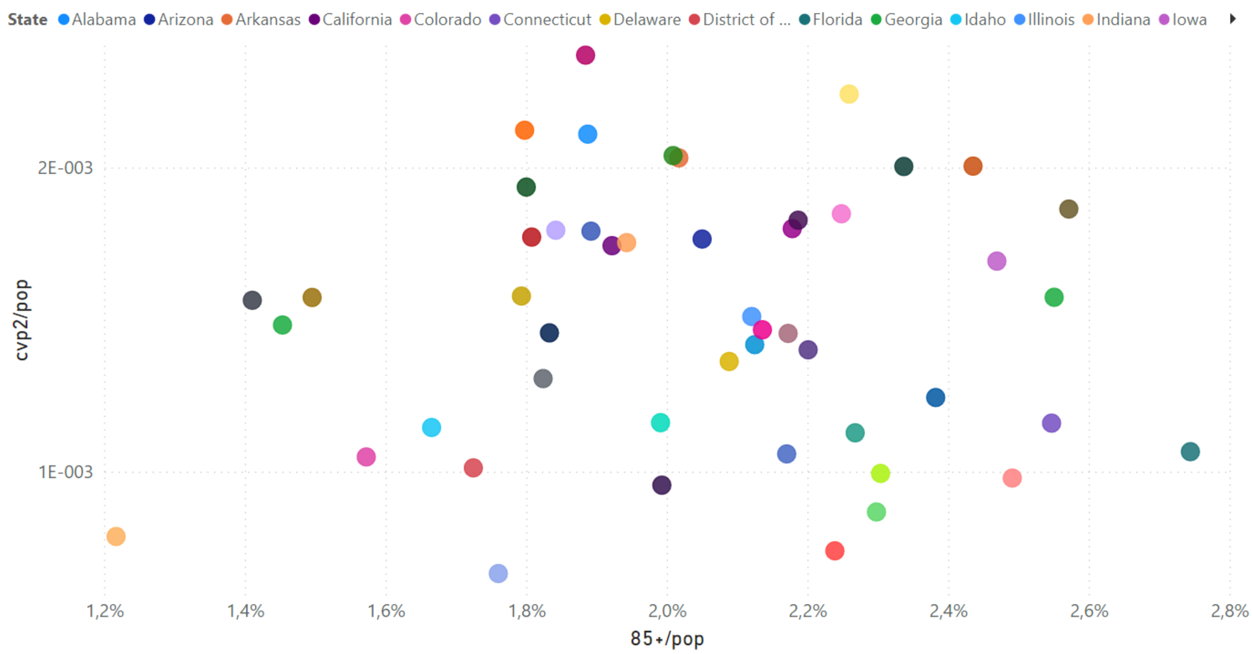


Figure 28c. cvp2/pop versus 85+/pop. Each point is for one continental USA state. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

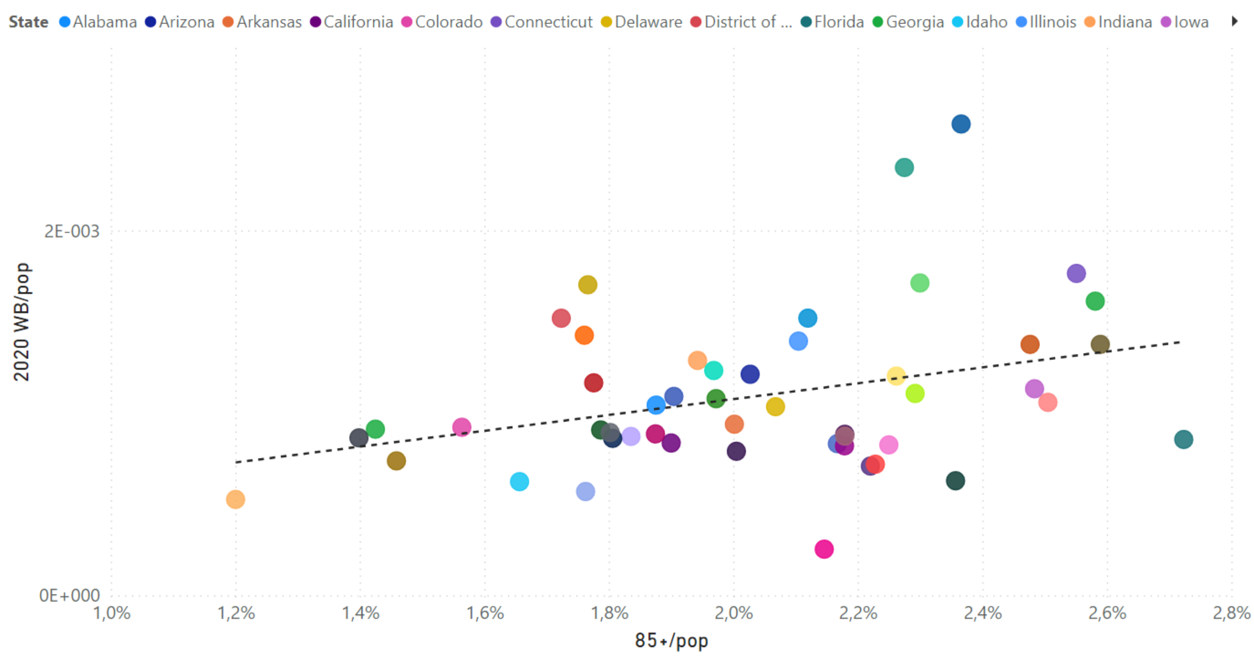


Figure 28d. WB/pop versus 85+/pop for cycle-year 2020. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

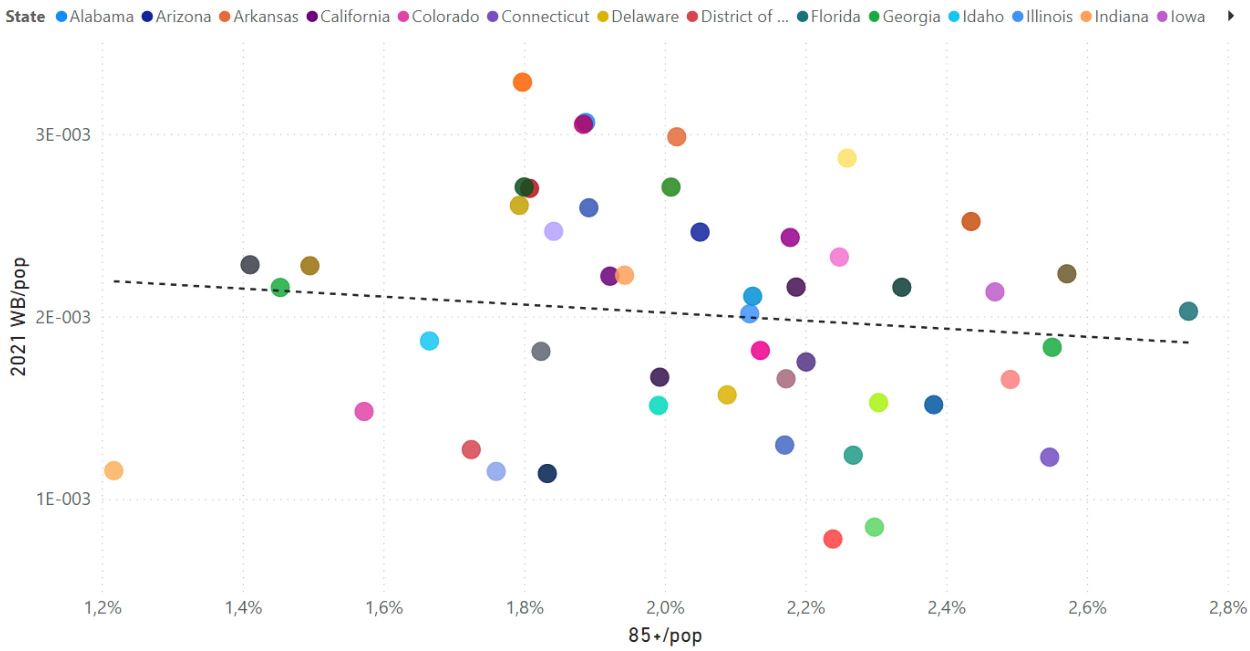


Figure 28e. WB/pop versus 85+/pop for cycle-year 2021. Each point is for one continental USA state. The trend line is meant merely to illustrate the correlation discussed in the text. It results from the usual least squares fit, using all the points in the graph. The colour-code of the 49 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

The same types of state-wise correlations for smp1 and cvp2 occur for other age groups also (data not shown). In summary, as follows.

- smp1/pop: pos-cor with -18/pop, neg-cor with 55-64/pop, neg-cor with 85+/pop
- cvp2/pop: pos-cor with -18/pop, neg-cor with 45-54/pop, neg-cor with 55-64/pop

Population density

The USA state-wise data offers a unique opportunity to examine the relation between population density ("popD") (number of inhabitants per unit surface area) and excess (above-SB) mortality, since popD varies by more than two orders of magnitude, from Wyoming to New Jersey.

Figure 29 shows WB/pop versus popD, for each pre-COVID-era cycle-year, 2014-2019. Here (Figure 29), there is no detectable, statistically significant, correlation between winter burden mortality (WB/pop) and popD, in any of the years studied.

Given the synchronous mortality patterns, state-to-state (Figures 10 and 11, for the pre-COVID-era cycle-years), and given present theoretical understanding of contagious disease transmission (Hethcote, 2000) (McCallum et al., 2001), our results (Figure 29) impose constraints on models of the phenomenon of seasonal mortality, and strongly suggest that the seasonal preponderance of viral respiratory diseases is not the result of transmission and spread by person-to-person “contact”.

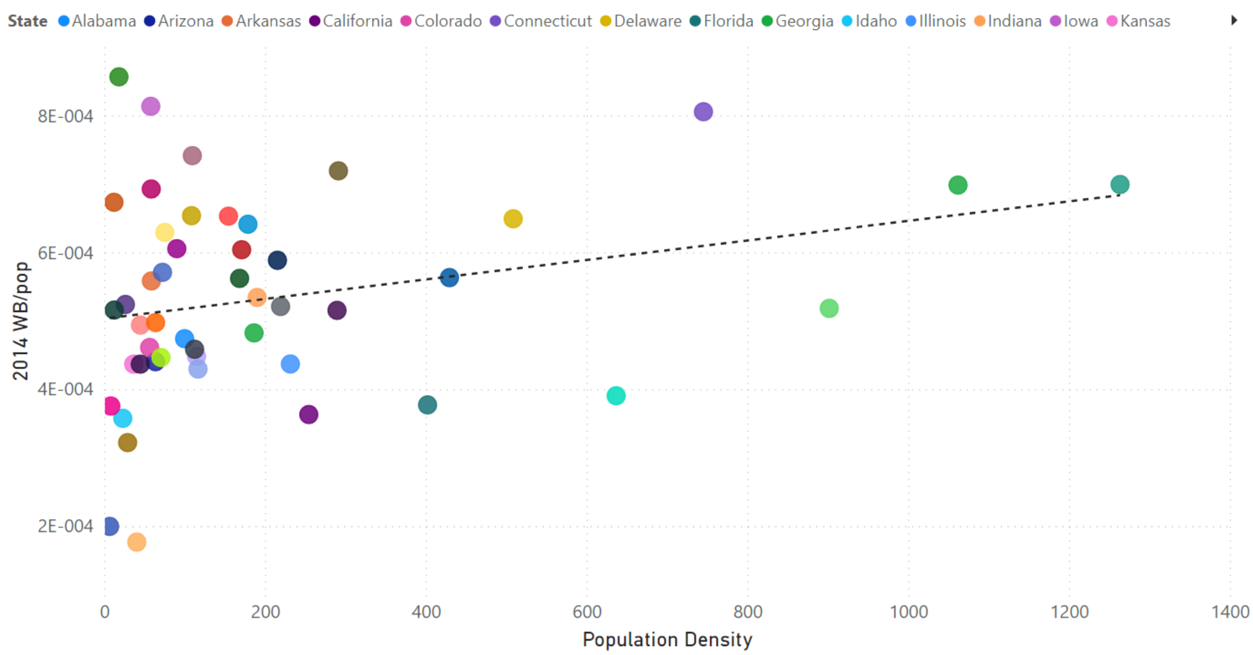


Figure 29a. WB/pop for cycle-year 2014 versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

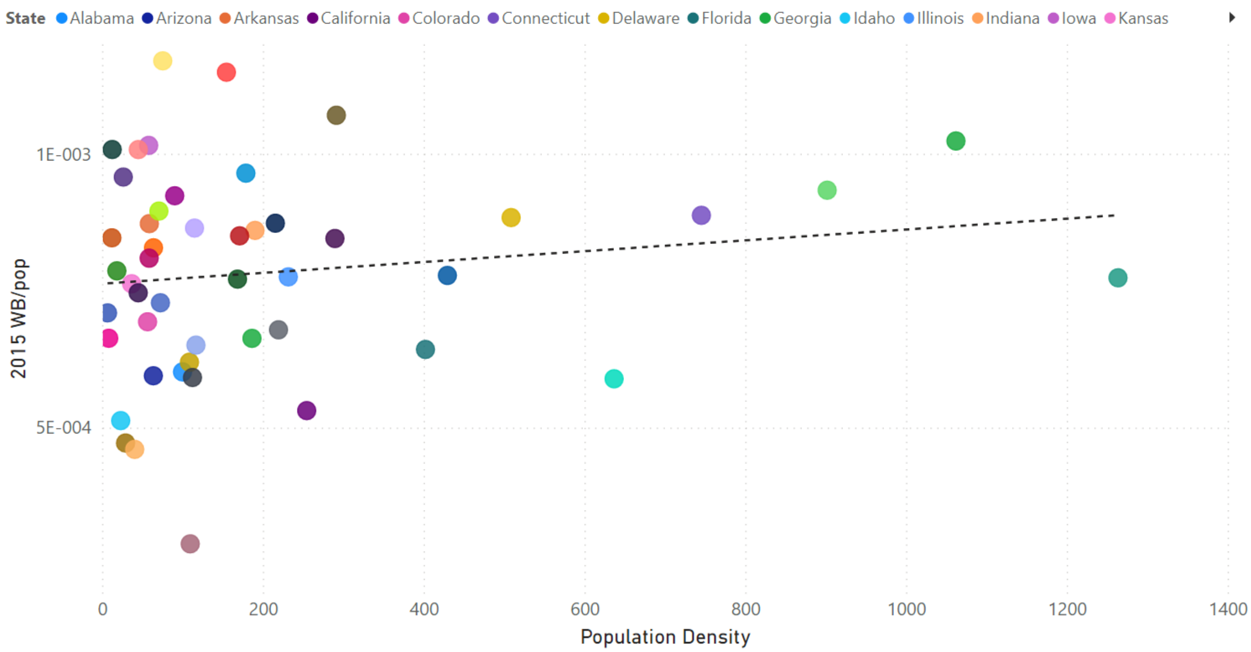


Figure 29b. WB/pop for cycle-year 2015 versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

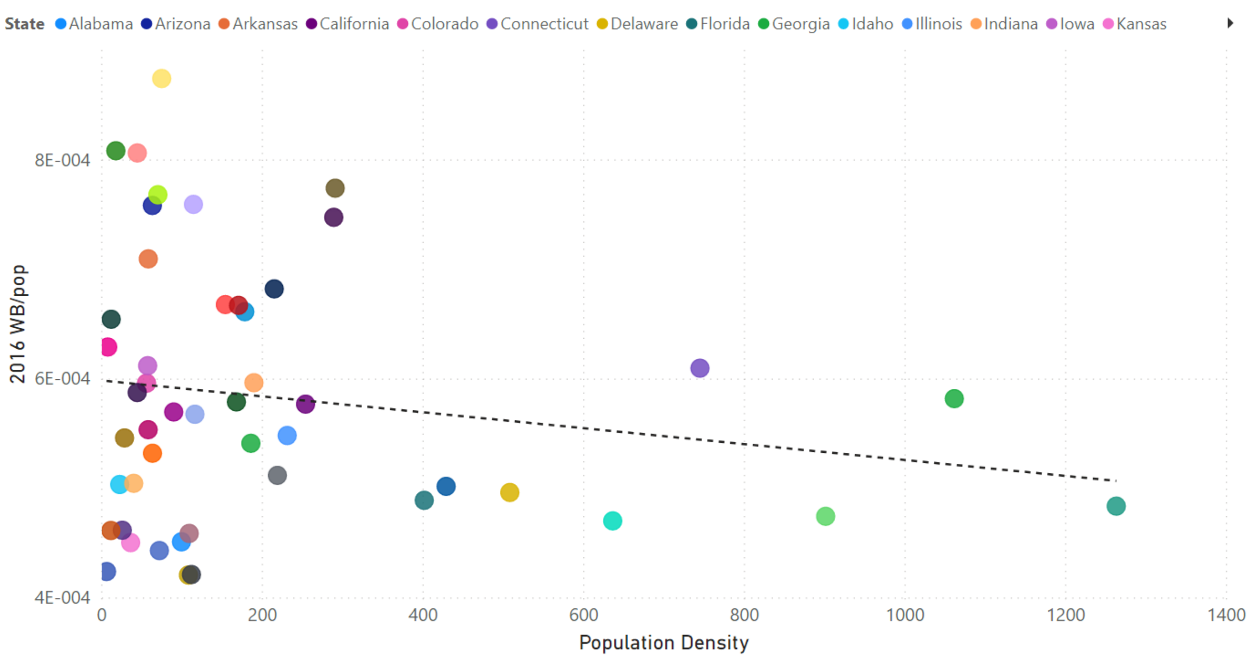


Figure 29c. WB/pop for cycle-year 2016 versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

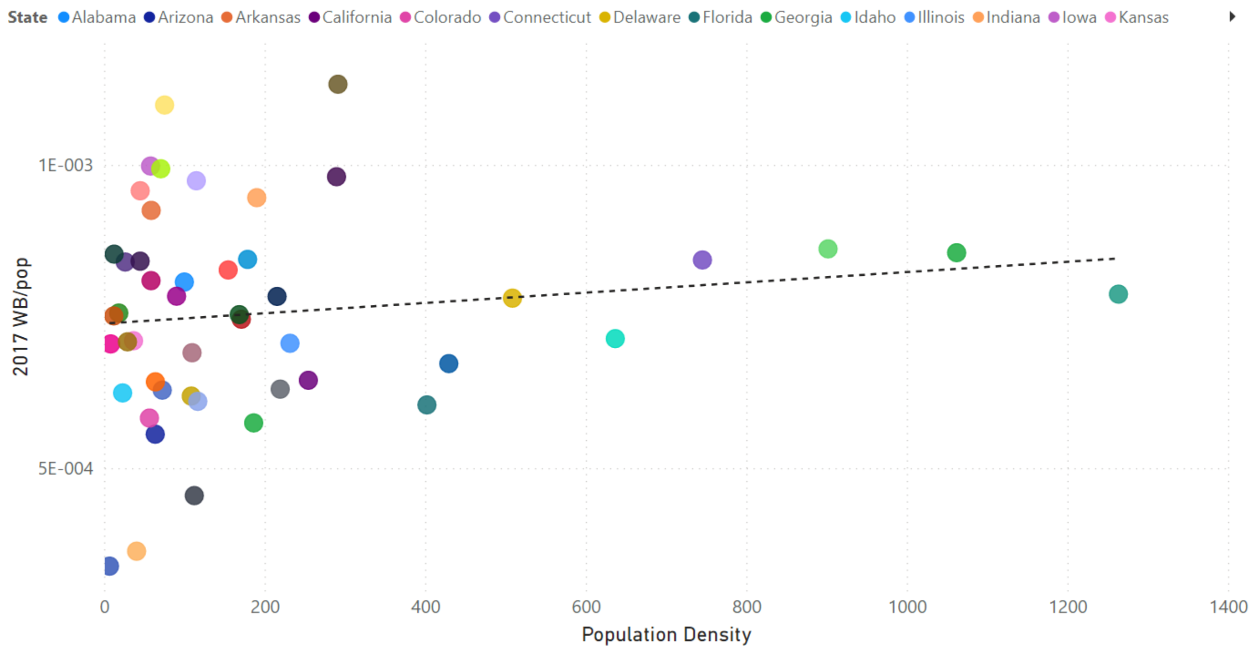


Figure 29d. WB/pop for cycle-year 2017 versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

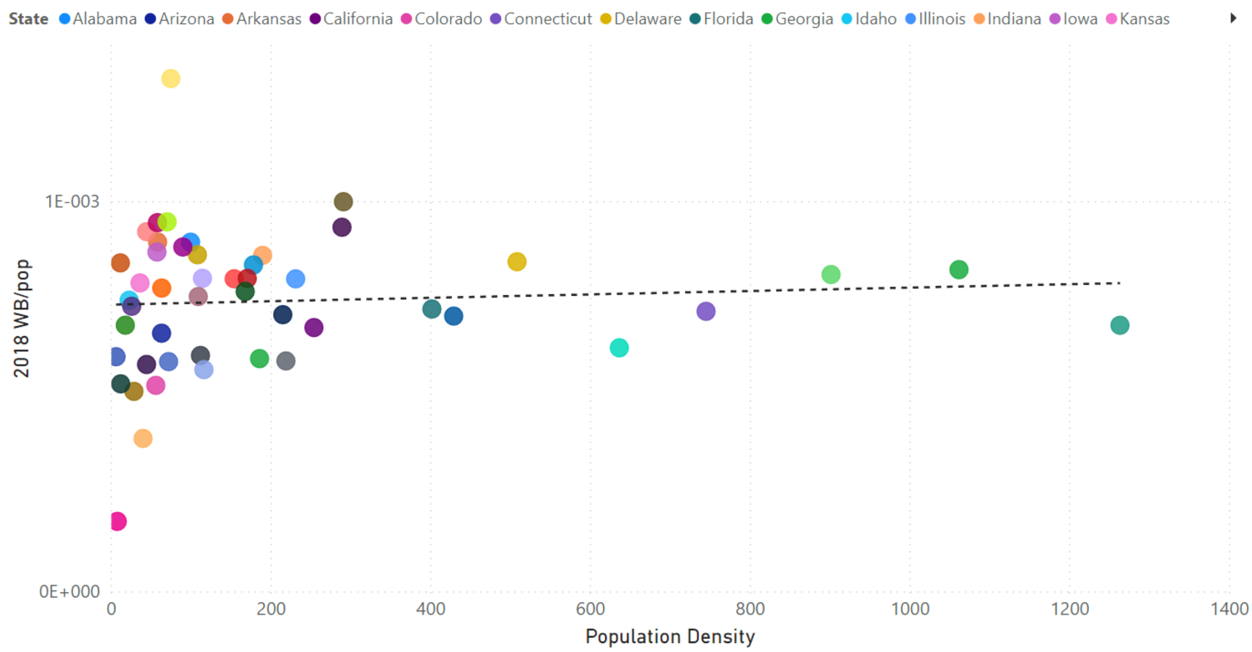


Figure 29e. WB/pop for cycle-year 2018 versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

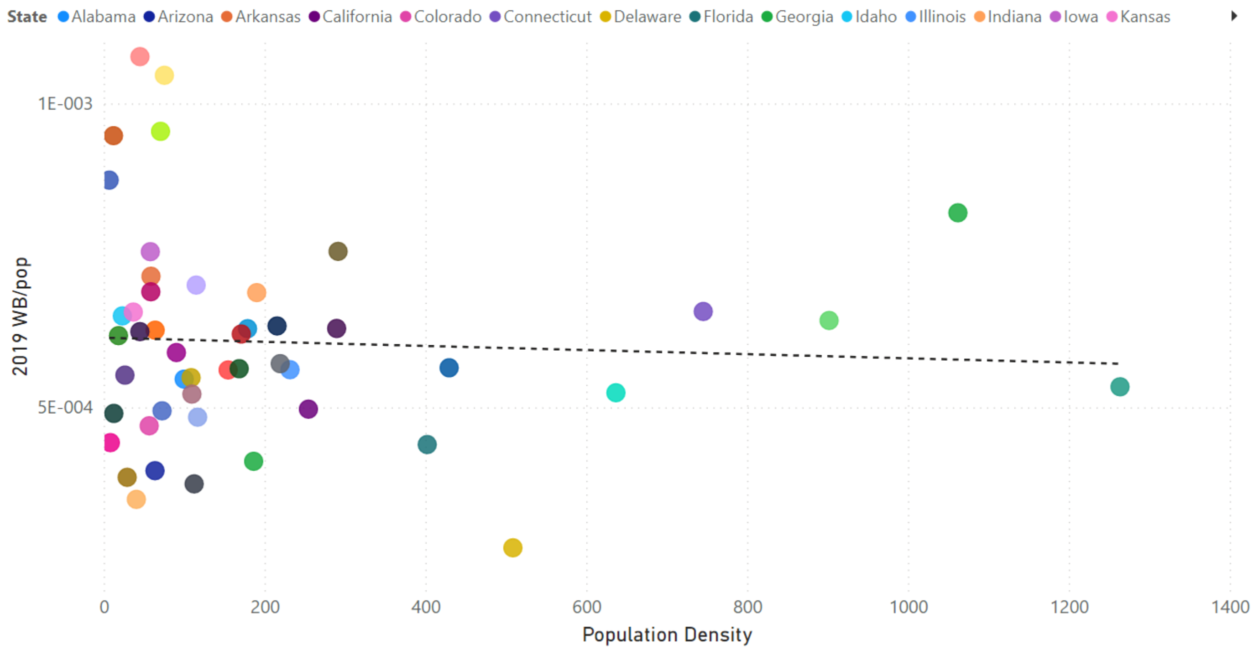


Figure 29f. WB/pop for cycle-year 2019 versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

This result (Figure 29) is in contrast to correlations observed for the COVID-era, where mortality has strong correlations and anti-correlations with popD. In the COVID-era, cvp1/pop has a large positive correlation with popD, although the New York outlier is significant, as shown in Figure 30a. While, on the other hand, both the summer-2020 excess (above-SB) mortality (smp1/pop) and the fall-winter-2020-2021 mortality (cvp2/pop) have anti-correlations with popD (Figures 30b and 30c, respectively). Correspondingly, the WB/pop versus popD has a large positive correlation for cycle-year 2020, with New York outlier (Figure 30d), and a strong negative (anti-)correlation for cycle-year 2021 (Figure 30e).

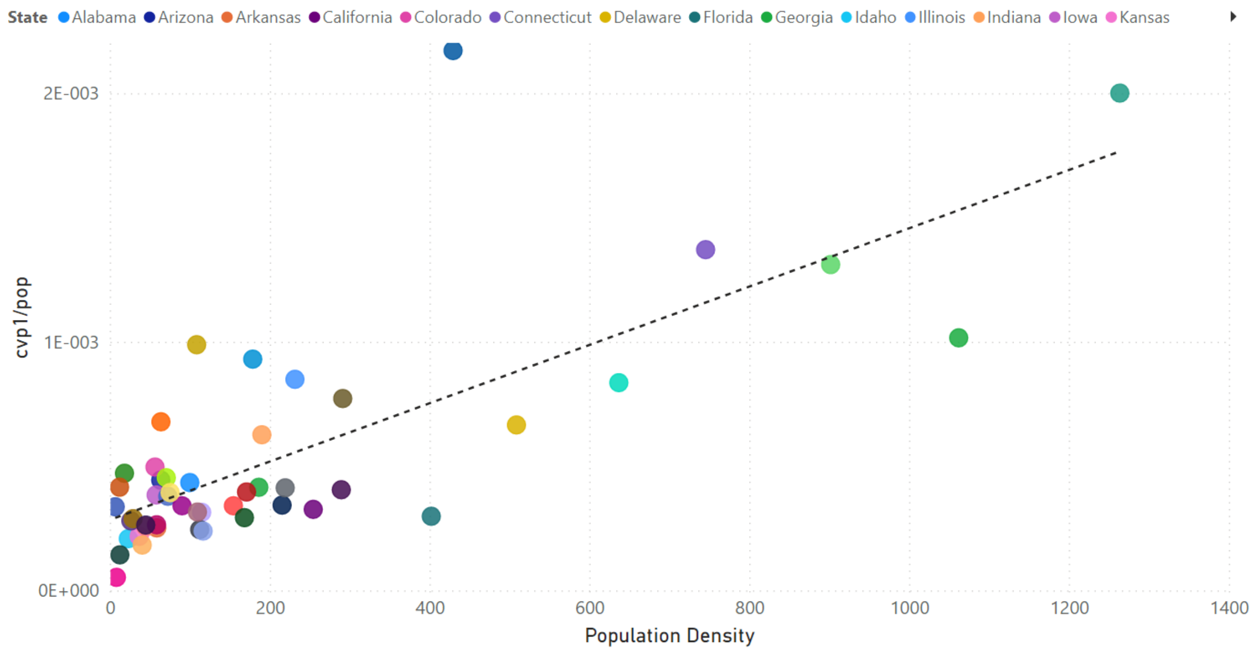


Figure 30a. cvp1/pop versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

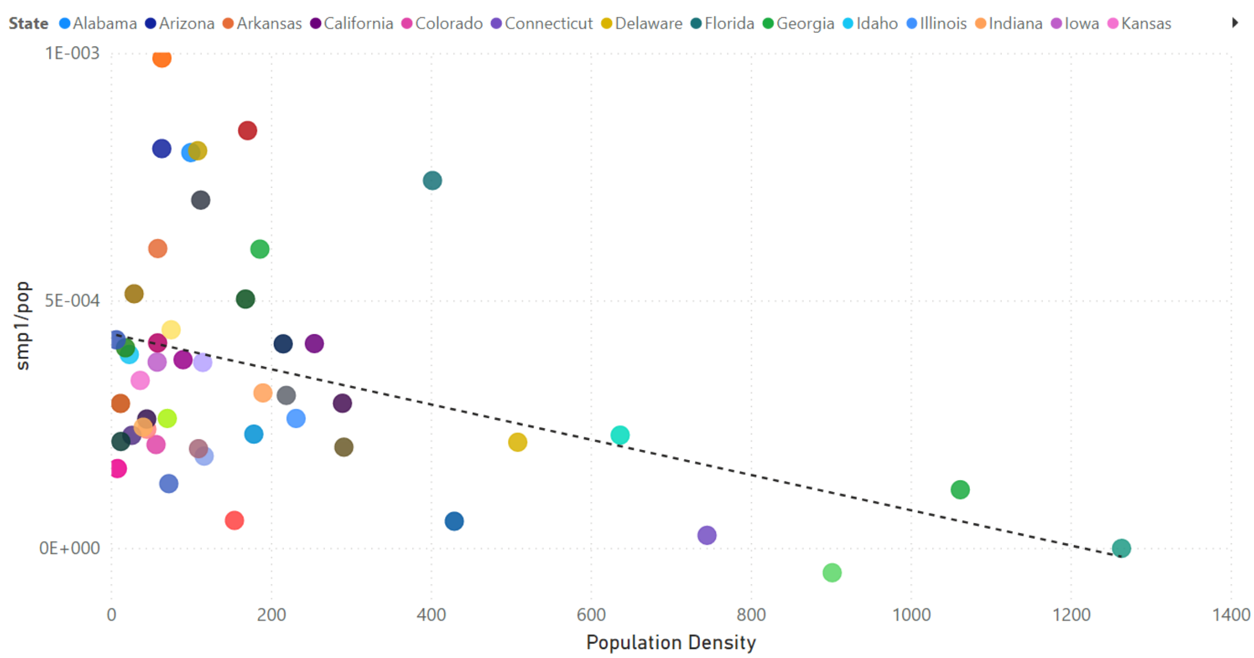


Figure 30b. smp1/pop versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

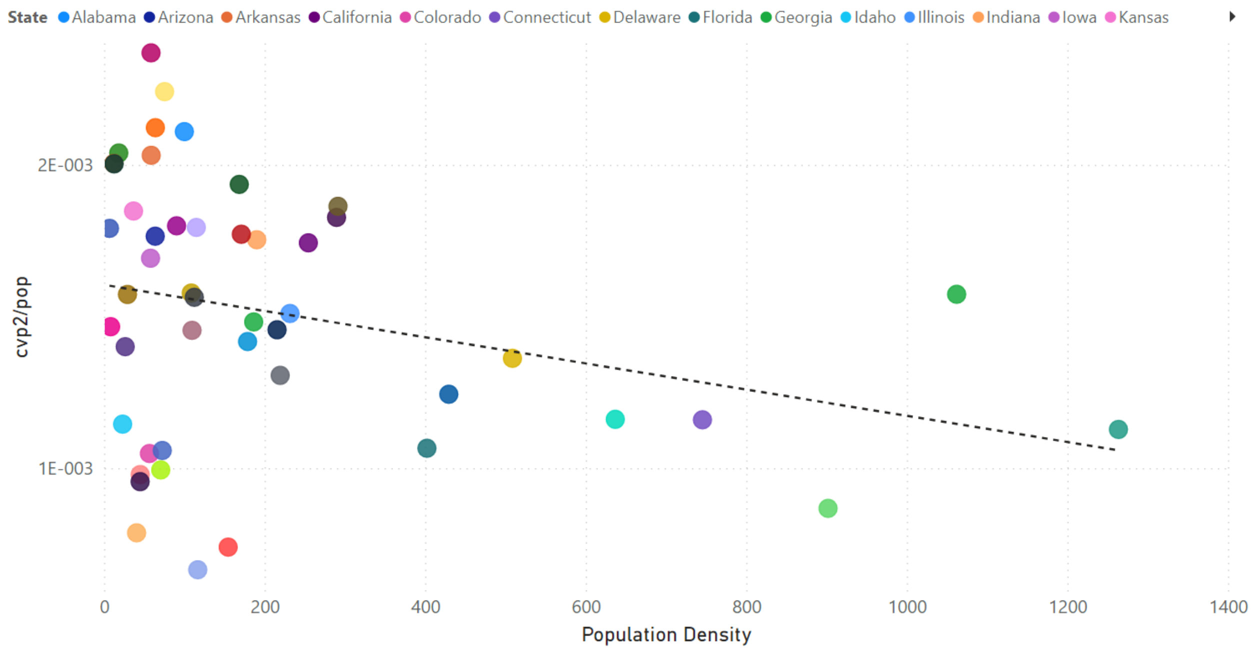


Figure 30c. cvp2/pop versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

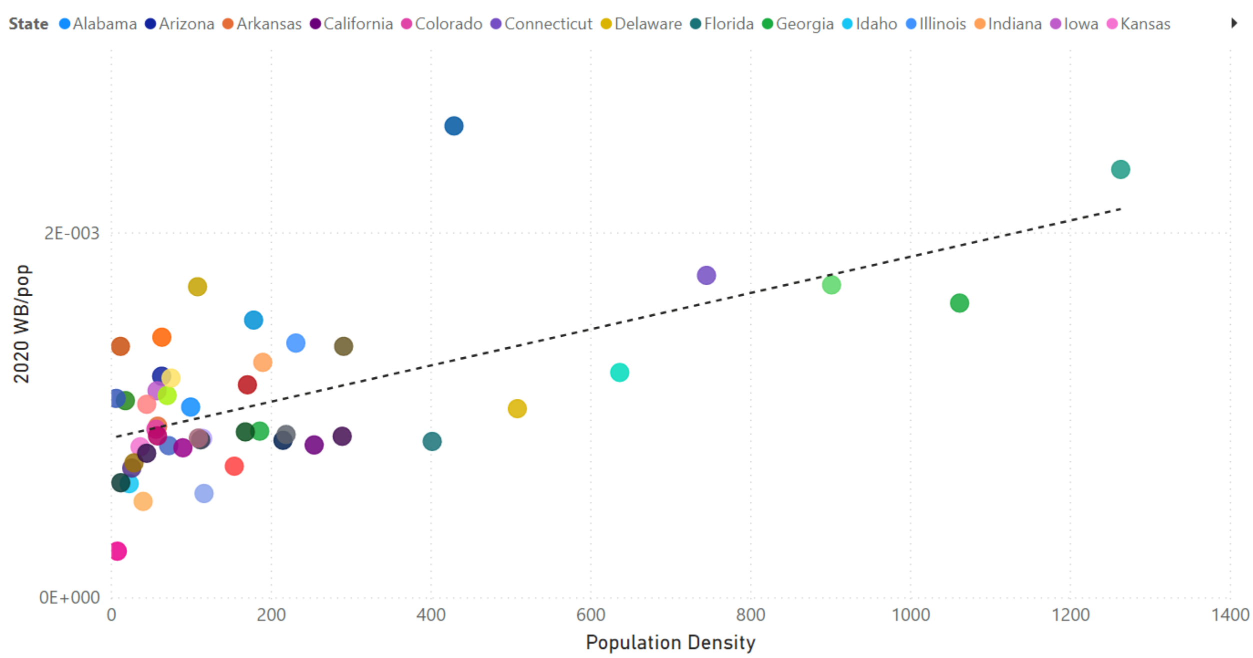


Figure 30d. WB/pop for cycle-year 2020 versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

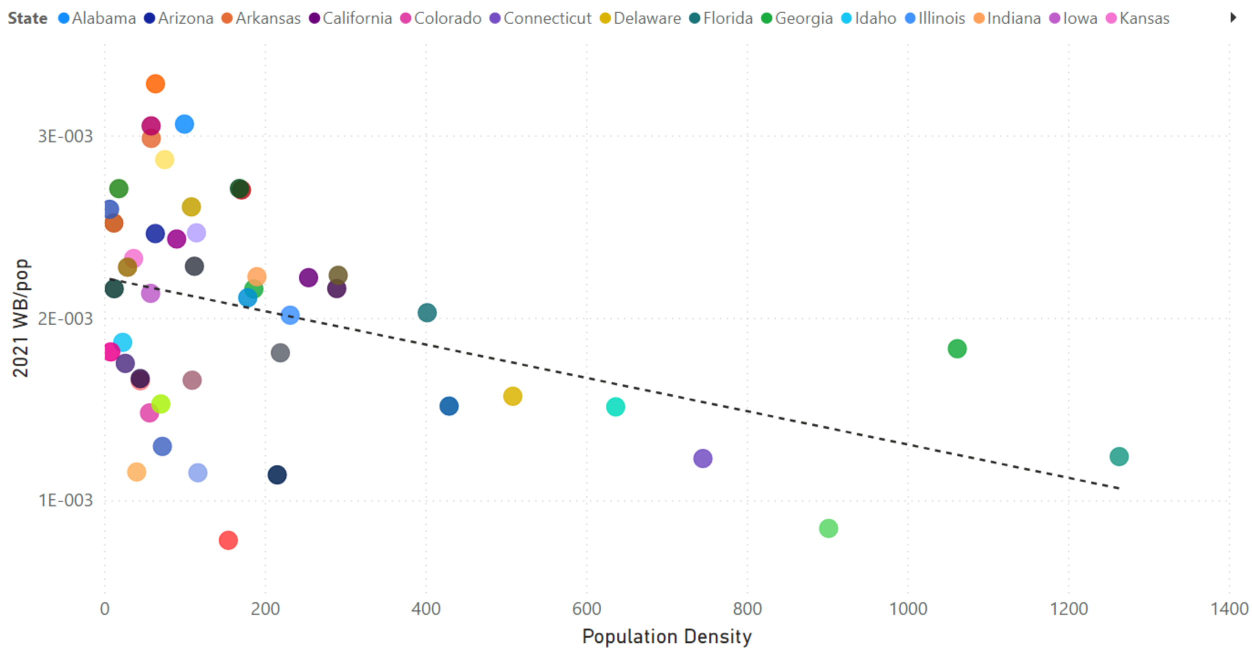


Figure 30e. WB/pop for cycle-year 2021 versus population density. Each point is for one continental USA state, excluding District of Columbia, which has an extreme density. The colour-code of the other 48 continental states is shown in section 2. Data were retrieved and calculations made as described in section 2.

We do not believe that a new virus causes the unprecedented correlations of mortality with popD, in the COVID-era. Rather, we interpret the results to mean that high-population-density states, with large urban centers would have had similar institutional structures and policy responses, generally different from those in low-population-density states. Also, the Southern states with large smp1 mortality due to climatic temperature, poverty and obesity are lower population-density states.

One pair of states, New York and Florida, strikingly demonstrates that population density in itself is not a controlling factor. Whereas these two states have essentially identical values of popD, they have diametrically opposed values of cvp1 mortality (Figure 30a), and, in the opposite order, of summer-2020 (smp1) mortality (Figure 30b).

Indeed, the correlations with popD in the COVID-era are an indication that the mortality is not the result of viral respiratory diseases, and rather that the mortality is tied to institutional, governmental, socio-economic and climatological differences.

All-cause mortality by week (ACM/w) by age group

The age dependencies of mortality in the pre-COVID and COVID-eras are shown more directly than only examining state-wise correlations, by examining ACM/w itself for the USA (no state-wise resolution is available) by age group, as follows.

We represent the ACM/w for the USA (Figure 5) by age group, for the two age groups 18-64 and 65+ ages, in Figure 32a. Here (Figure 32a), we have multiplied the ACM/w for the 18-64 years age group by a factor sufficient to make the ACM/w equal to that for the 65+ years age group, in the summer-2014 trough. This is equivalent to multiplying the population of the 18-64 years age group until the deaths per week are equal to the deaths per week in the 65+ years age group, in the summer-2014 trough. This is done to better visualize and compare the relative seasonal changes in mortality between the two age groups.

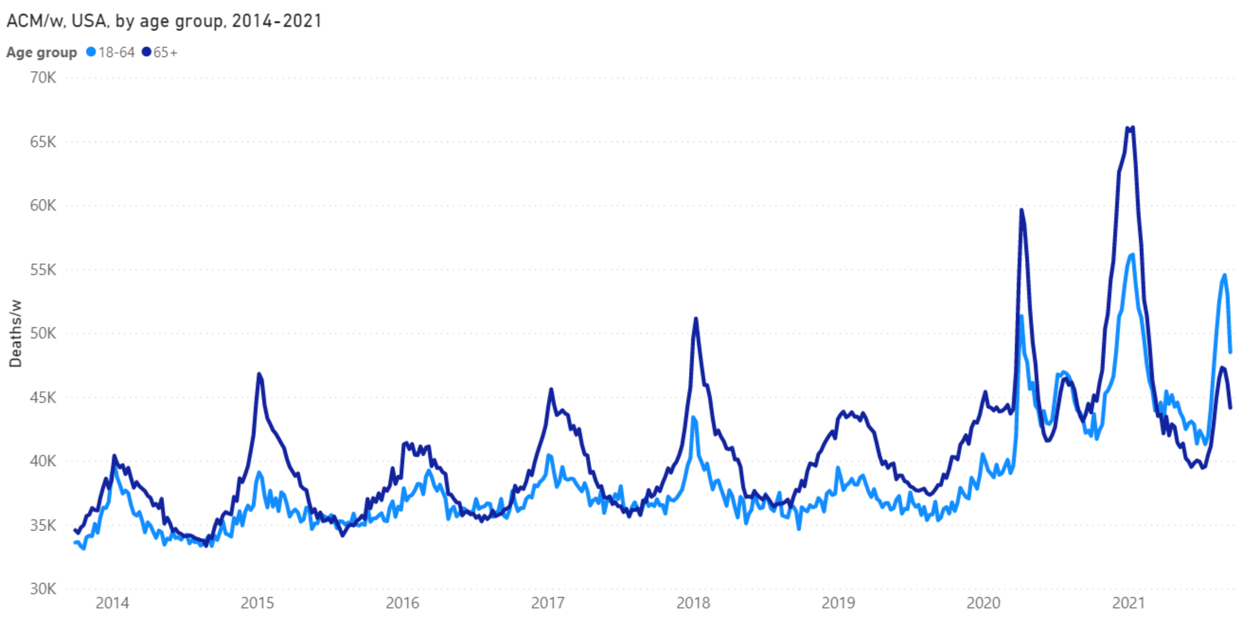


Figure 32a. All-cause mortality by week in the USA for the 18-64 and 65+ years age groups (light blue and dark blue lines, respectively), from 2014 to 2021. The ACM/w for the 18-64 years age group is rescaled (multiplied), as explained in the text, to make the number of deaths per week of both age groups equal in the summer-2014 trough, for comparison purposes. Data are displayed from week-40 of 2013 to week-37 of 2021 for the whole

continental USA, including Alaska and Hawaii. Data were retrieved from CDC (CDC, 2021a), as described in Table 1.

Figure 32a shows that, in the pre-COVID-era, the elderly group (65+ years) is always approximately 2-3 times more susceptible to the additional challenges and stress of winter than the younger group (18-64 years). This rule is not followed in the COVID-era. In the COVID-era, the relative summer-2020 and summer-2021 mortalities are greater for the younger age group than for the elderly group (Figure 32a), which is reversed compared to known age-dependent vulnerability to dying from viral respiratory diseases.

This reversal in the COVID-era is more explicitly illustrated in Figure 32b, which shows the difference by week of the two curves depicted in Figure 32a.

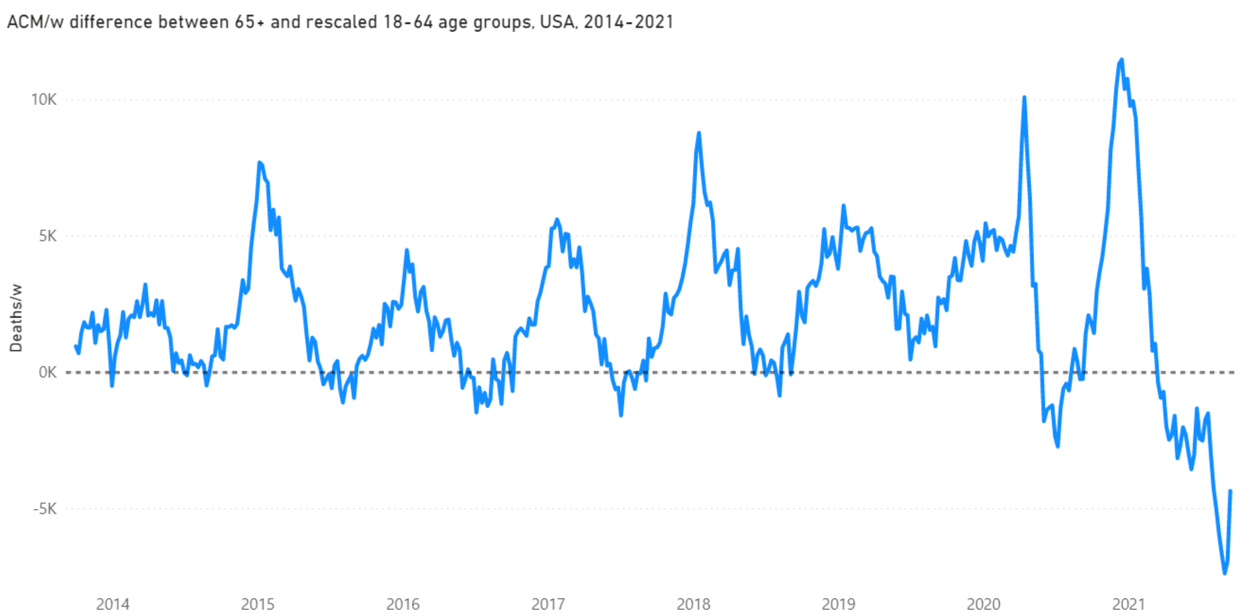


Figure 32b. Difference in all-cause mortality by week in the USA between the 65+ years and the rescaled 18-64 years age groups, from 2014 to 2021. The ACM/w for the 18-64 years age group was rescaled (multiplied), as explained in the text, to make the number of deaths per week of both age groups equal in the summer-2014 trough, for comparison purposes. Data are displayed from week-40 of 2013 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The dashed line emphasizes the zero. Data were retrieved from CDC (CDC, 2021a), as described in Table 1.

Here (Figure 32b), we see that the younger age group (18-64 years) has moderately more (rescaled) deaths in summer-2020, and significantly more (rescaled) deaths in summer-2021. Two possible interpretations come to mind: either the integrated cumulative long-term stress from the government measures takes longer to affect more tolerant younger individuals than older individuals, or the massive vaccination campaign administered between the two summers (Figure 31, below) has had a disproportionate negative impact on the younger age group.

A more detailed examination of the COVID-era is possible thanks to more age-group resolution being publicly available for that time period (CDC, 2021b), at the national level (not state-resolved), as follows. A selection of these data is shown in Figure 33.

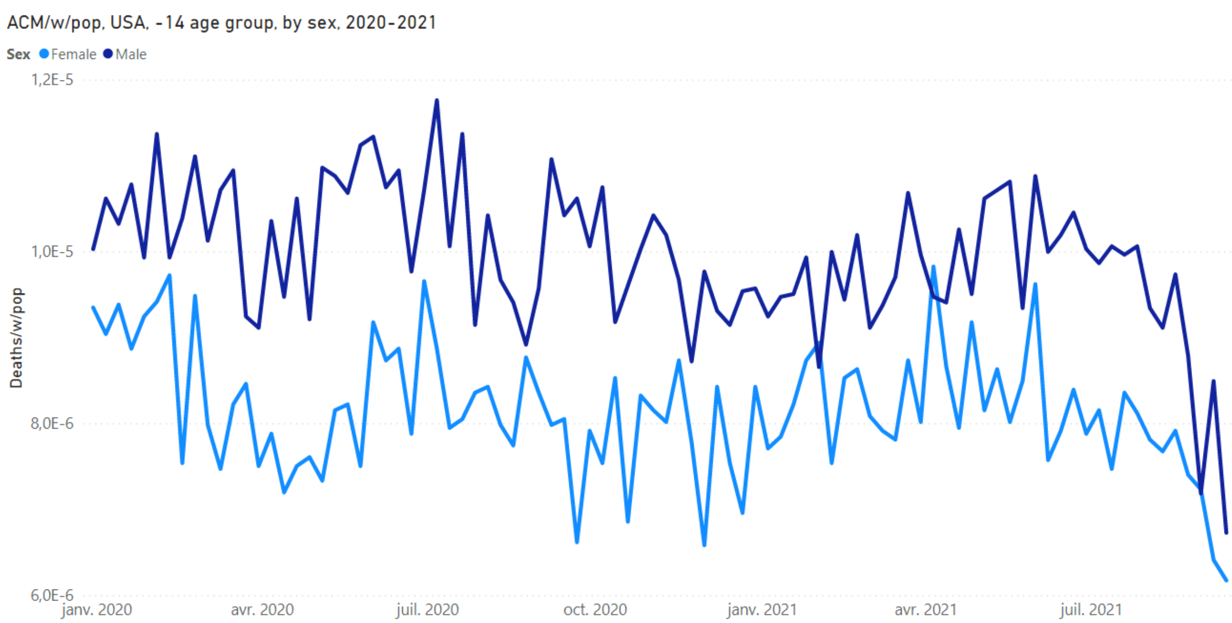


Figure 33a. All-cause mortality by week normalized by population for the USA for the 14 years and less ("-14 years") age group, for each of both sexes, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The population used for normalization is the population of the specific age group and sex. ACM data were retrieved from the CDC (CDC, 2021b) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021b), as described in Table 1.

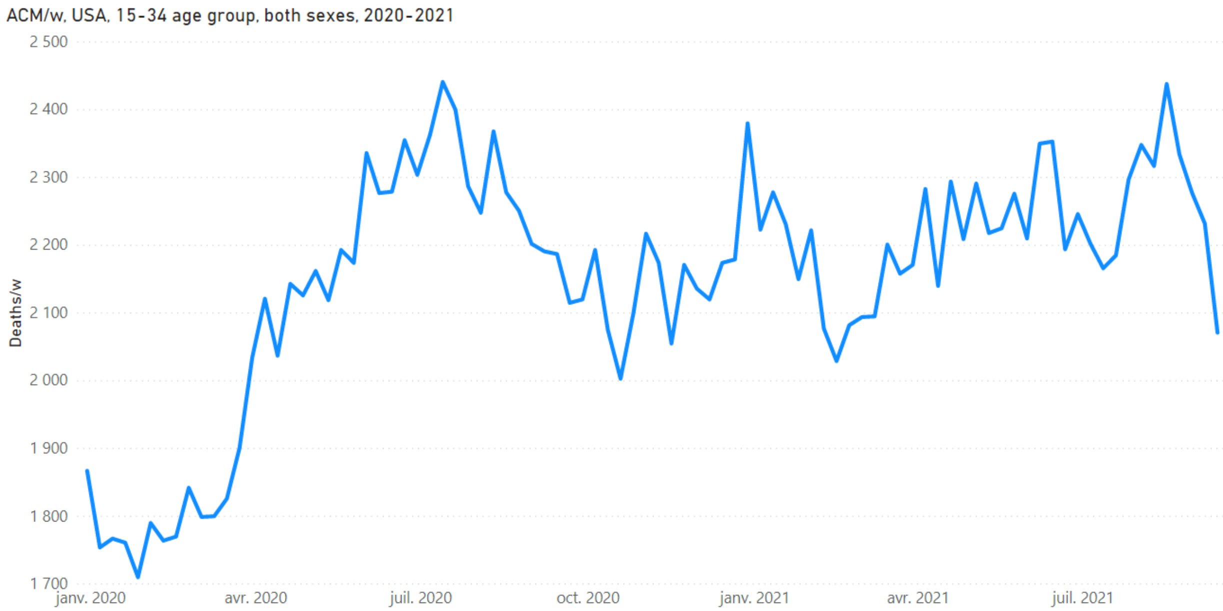


Figure 33b. All-cause mortality by week for the USA for the 15-34 years age group, both sexes, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The population used for normalization is the population of the specific age group. ACM data were retrieved from the CDC (CDC, 2021b) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021b), as described in Table 1.

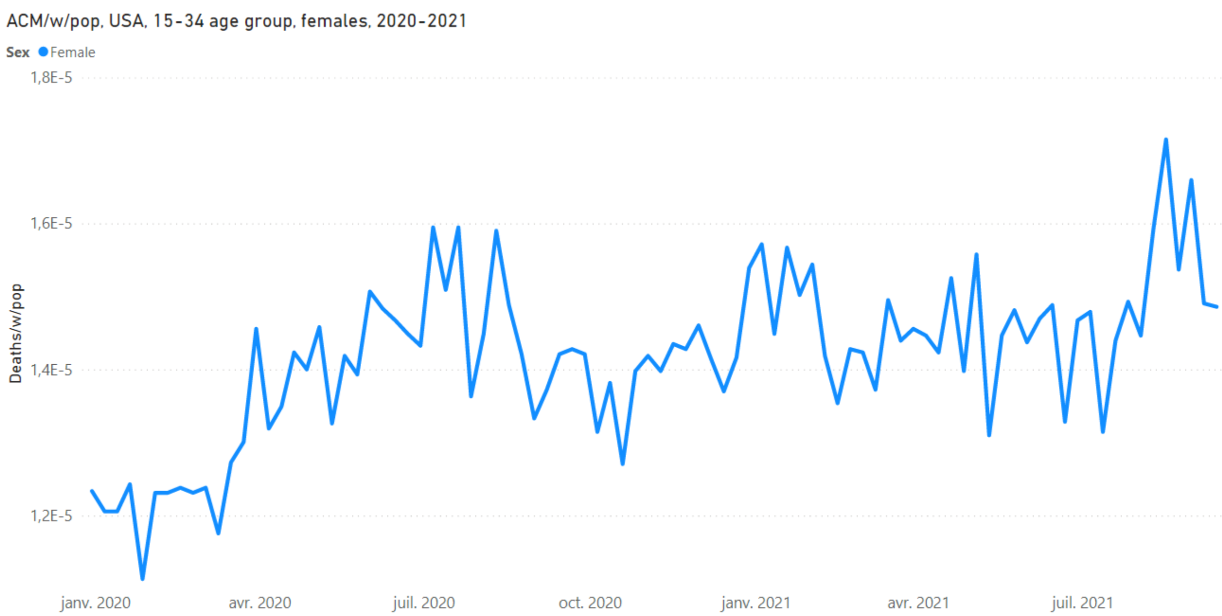


Figure 33c. All-cause mortality by week normalized by population for the USA for females of the 15-34 years age group, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The population

used for normalization is the population of the specific age group and sex. ACM data were retrieved from the CDC (CDC, 2021b) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021b), as described in Table 1.

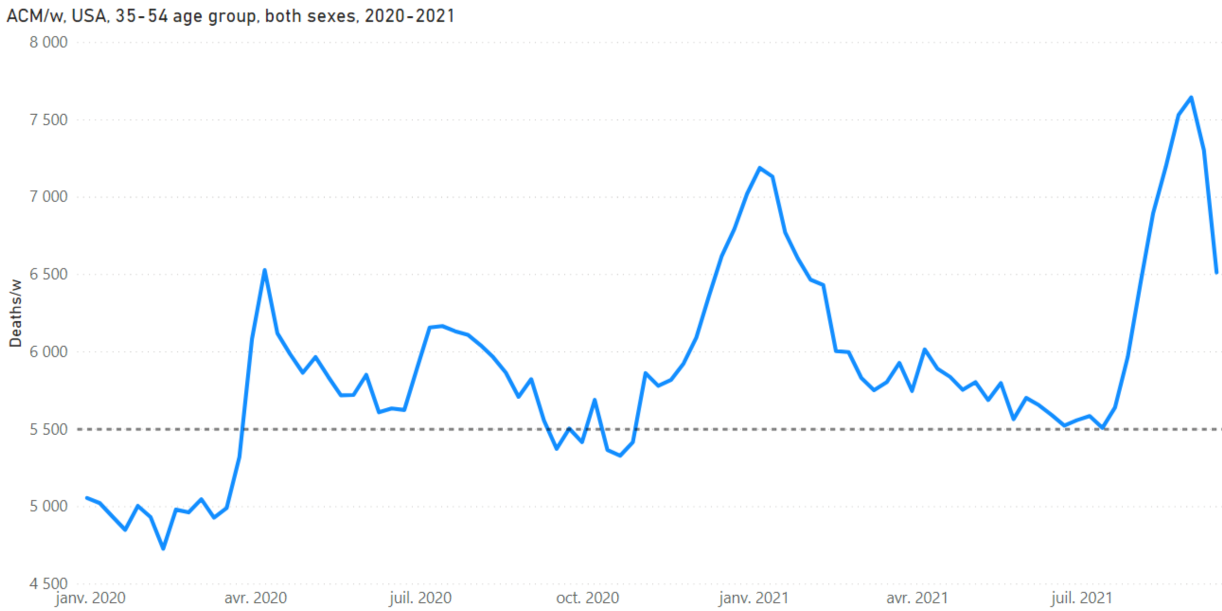


Figure 33d. All-cause mortality by week for the USA for the 35-54 years age group, both sexes, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The population used for normalization is the population of the specific age group. ACM data were retrieved from the CDC (CDC, 2021b) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021b), as described in Table 1. The horizontal line at “5 500” is a visual aide of the plateau of mortality discussed in the text.

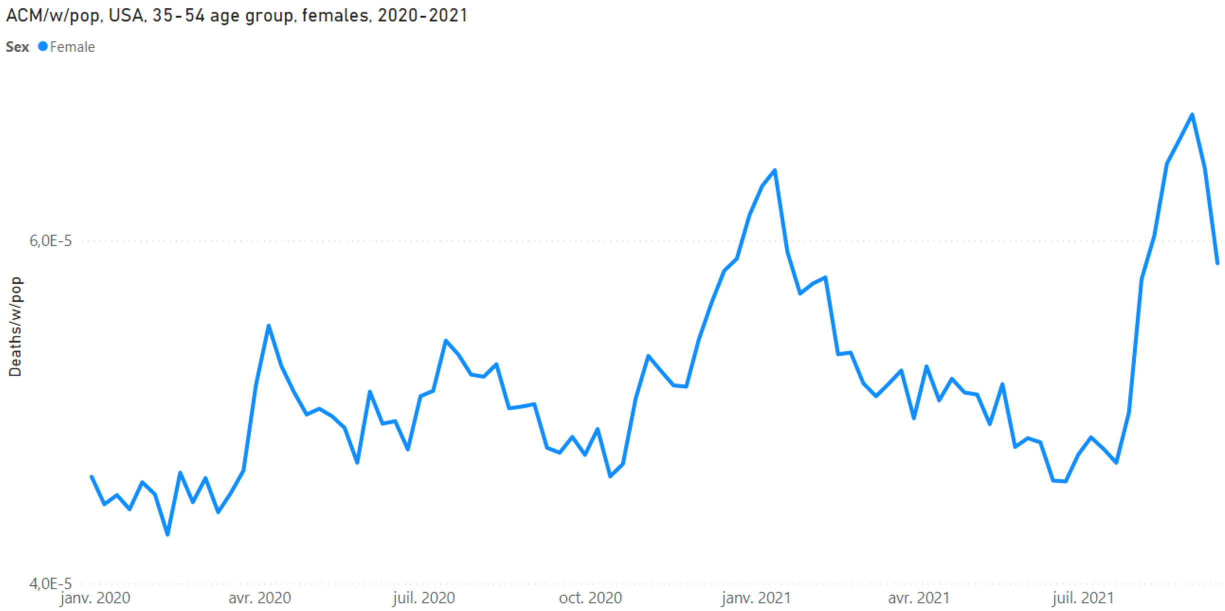


Figure 33e. All-cause mortality by week normalized by population for the USA for females of the 35-54 years age group, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The population used for normalization is the population of the specific age group and sex. ACM data were retrieved from the CDC (CDC, 2021b) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021b), as described in Table 1.

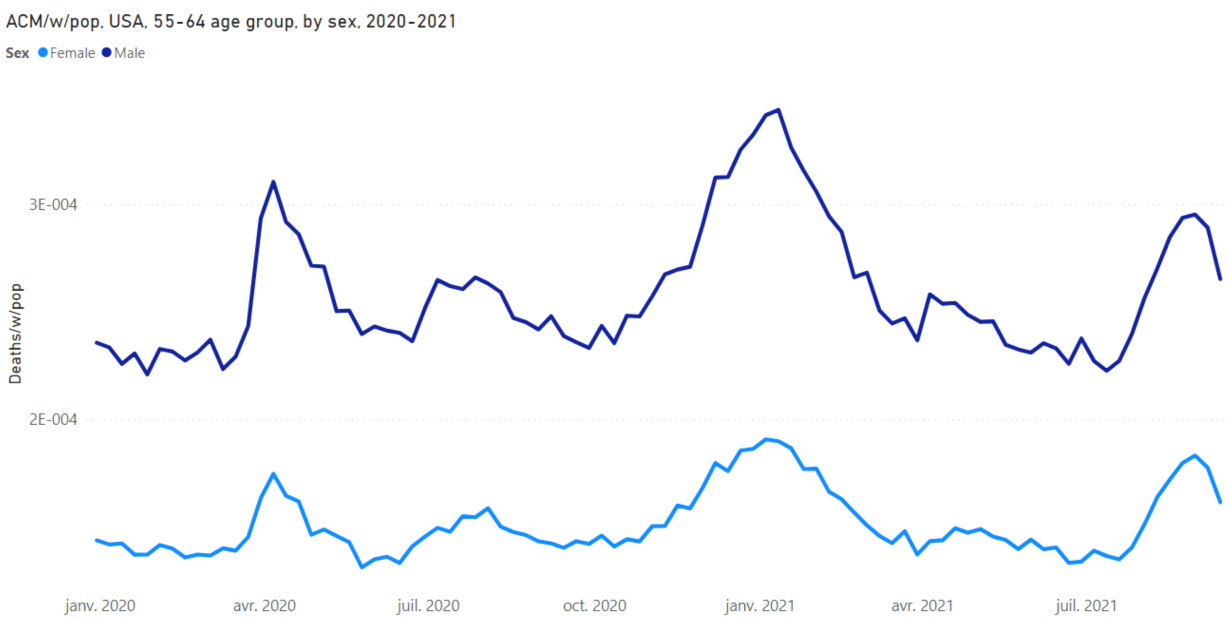


Figure 33f. All-cause mortality by week normalized by population for the USA for the 55-64 years age group, for each of both sexes, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii.

The population used for normalization is the population of the specific age group and sex. ACM data were retrieved from the CDC (CDC, 2021b) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021b), as described in Table 1.

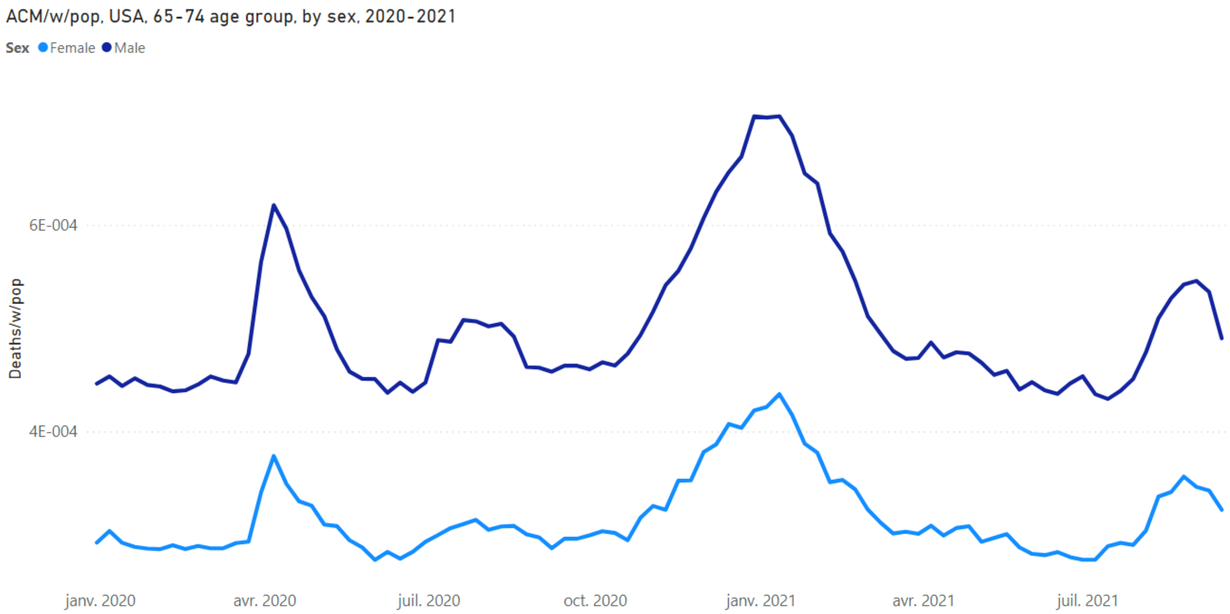


Figure 33g. All-cause mortality by week normalized by population for the USA for the 65-74 years age group, for each of both sexes, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The population used for normalization is the population of the specific age group and sex. ACM data were retrieved from the CDC (CDC, 2021b) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021b), as described in Table 1.

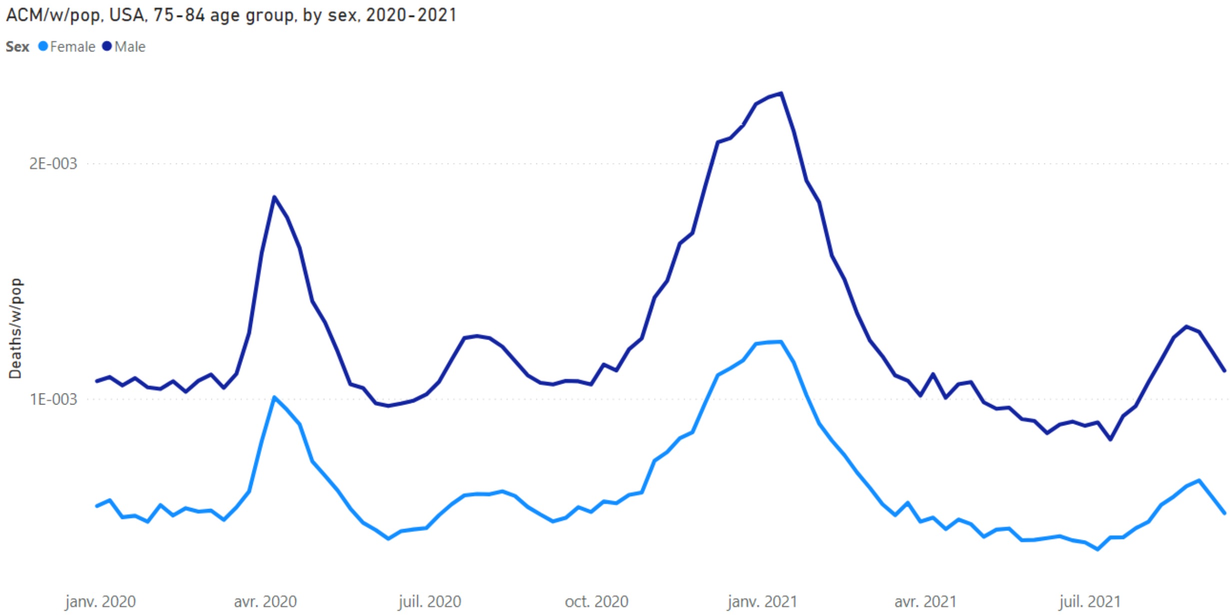


Figure 33h. All-cause mortality by week normalized by population for the USA for the 75-84 years age group, for each of both sexes, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The population used for normalization is the population of the specific age group and sex. ACM data were retrieved from the CDC (CDC, 2021b) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021b), as described in Table 1.

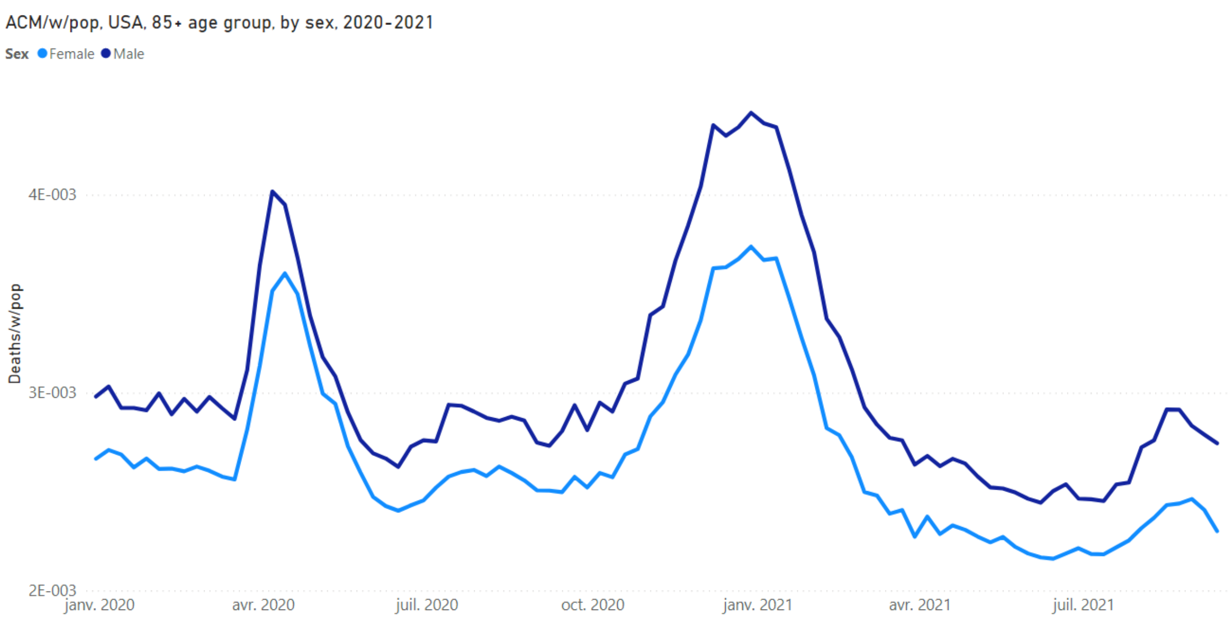


Figure 33i. All-cause mortality by week normalized by population for the USA for the age group 85 years and older ("85+ years"), for each of both sexes, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021 for the whole continental USA, including

Alaska and Hawaii. The population used for normalization is the population of the specific age group and sex. ACM data were retrieved from the CDC (CDC, 2021b) and population data were retrieved from the US Census Bureau (US Census Bureau, 2021b), as described in Table 1.

Figure 33 shows the following:

- (Figure 33a) In the -14 years age group there is no evidence for any summer/winter seasonality, or any COVID-era anomalies. The ACM/w/pop is essentially flat over the time period. Young (-14 years) residents of the USA are essentially not killed by viral respiratory diseases or COVID-19 or any cause of death having a strong seasonal variation in its effect.
- (Figures 33b and 33c) Figure 33b shows that the onset of the COVID-era (March 2020) is associated with an increase in deaths of 15-34 year olds to a new plateau in ACM/w (approximately 400 more deaths per week), which does not return to normal over the period studied. The rise to a COVID-era plateau of increased mortality occurs for both males and females (Figure 33c).
- (Figures 33d and 33e) The 35-54 years age group, like the 15-34 years age group, also experiences a high essentially uniform baseline plateau of mortality, which does not return to normal values over the period studied, but the ACM/w for this age group (35-54 years) also shows distinct cvp1, smp1, cvp2 and smp2 features superposed on the said plateau. This age group (35-54 years) has a disproportionately large smp2 feature (summer-2021 mortality), compared to the other features, and using the smp1 and cvp2 features as references, which holds for both males and females (Figure 33e).
- (Figures 33f, 33g, 33h and 33i) The age groups 55-64, 65-74, 75-84 and 85+ years do not exhibit the COVID-era increased baseline plateau mortalities seen in the 15-34 and 35-54 years age groups. Summer mortality for both 2020 (smp1) and 2021 (smp2) monotonically decrease in relative magnitude, compared to the cvp1 and cvp2 features, as age increases in the sequence 55-64, 65-74, 75-84 and 85+ years.

The results regarding dependence of mortality on state-to-state age structure of the population (Figures 27 and 28) show that the summer-2020 excess (above-SB) deaths

were not predominantly due to viral respiratory diseases, and impacted younger people. Likewise, we deduce that the excess (above-SB) deaths in fall-winter-2020-2021 must predominantly be due to causes other than viral respiratory diseases, and impacted younger people. The inferred impacts on younger residents are corroborated by the age-group-specific mortalities at the national level (Figures 32 and 33).

Comparing all-cause excess mortality and COVID-assigned mortality

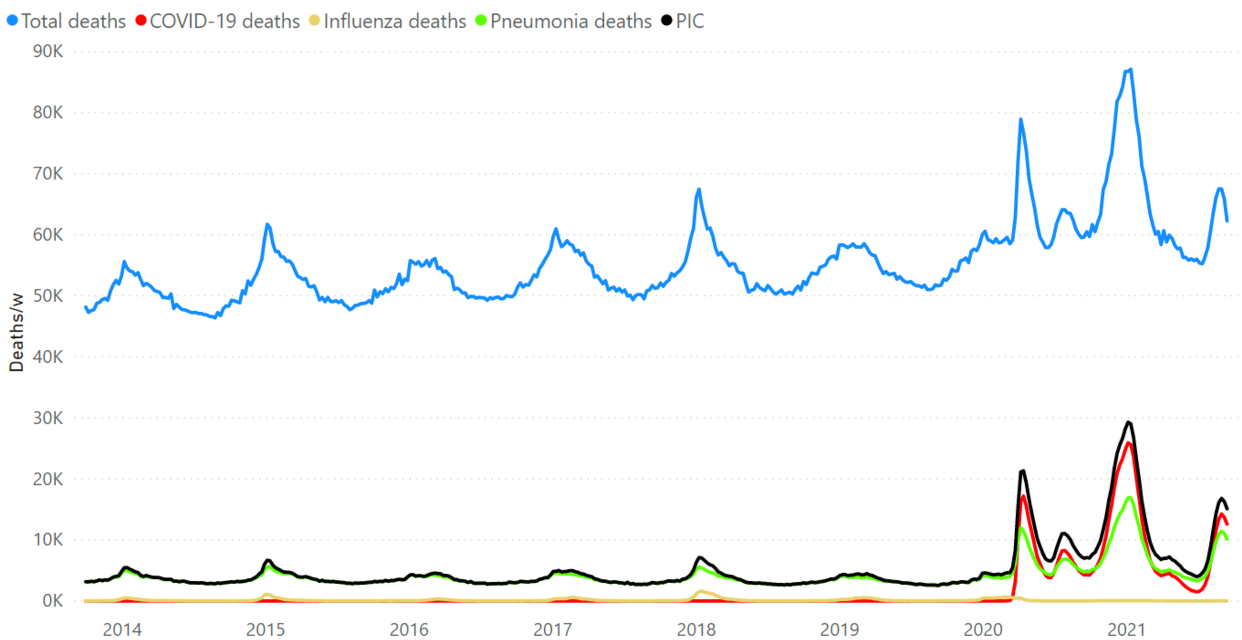
COVID-19-assigned deaths cannot be trusted to be deaths actually caused by COVID-19 (Borger et al., 2021). Furthermore, it is likely that the COVID-19 assignation of cause of death captures far too many deaths (Elsoukkary et al., 2021). Nonetheless, we can compare the total number of COVID-19-assigned deaths in the USA to excess (above-SB) all-cause mortality.

For the two cycle-years 2020 and 2021 (July 2019 to July 2021), the total WB is 1.071 M deaths, compared to total CDC-reported COVID-assigned deaths up to July 2021 (up to the last week of the 2021 cycle-year, week-30 of 2021, which is the week of 26 July 2021) equal to 613 K deaths (CDC, 2021a, as described in the Table 1). Both numbers include Alaska and Hawaii. This leaves some 458 K above-SB deaths, up to July 2021, which are not accounted for by COVID-19 according to the relevant CDC statistics.

The difference of 458 K deaths, if the COVID-19-assignations could be trusted (they cannot), would be consistent with a large number of deaths (458 K) of younger residents whose deaths are not assigned to COVID-19 (Kostoff et al., 2021; their Figure 1). In addition to our results, above, Jacobson and Jokela (2021) also found that large numbers of individuals, too young to have died from COVID-19, died in the COVID-era.

To examine this difference (458 K deaths) more closely, we compare the all-cause mortality by week to assigned-cause deaths by week for pneumonia (P), influenza (I)

and COVID-19 (C), reported by the CDC (2021a), in Figure 34; for 2014-2021 (Figure 34a) and on the expanded scale 2019-2021 (Figure 34b). PIC by week is also shown, which is the deaths assigned by the CDC as “pneumonia, influenza, and/or COVID-19”, which means that the death certificate includes pneumonia and/or influenza and/or COVID-19 listed as cause(s) of death.



Figures 34a. All-cause (blue), COVID-19 (red), influenza (yellow), pneumonia (green) and PIC (black) mortality by week for the USA from 2014 to 2021. Data are displayed from week-40 of 2013 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. PIC is the deaths assigned to pneumonia and/or influenza and/or COVID-19. ACM and cause-assigned deaths data were retrieved from CDC (CDC, 2021a) as described in Table 1.

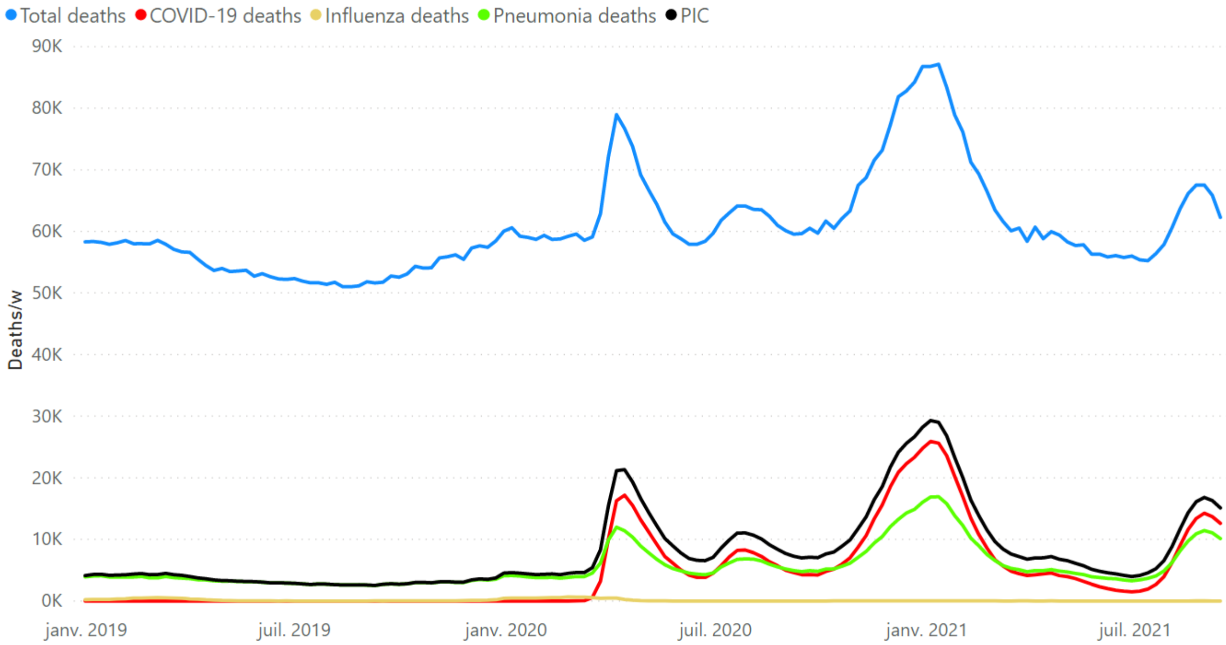


Figure 34b. All-cause (blue), COVID-19 (red), influenza (yellow), pneumonia (green) and PIC (black) mortality by week for the USA from 2019 to 2021. Data are displayed from week-1 of 2019 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. PIC is the deaths assigned to pneumonia and/or influenza and/or COVID-19. ACM and cause-assigned deaths data were retrieved from CDC (CDC, 2021a) as described in Table 1.

We interpret the similarity in patterns of temporal variation between CDC-reported weekly COVID-19-assigned or PIC deaths and the all-cause mortality (ACM/w) as arising because many or most of the COVID-19-assigned deaths are drawn from our above-SB deaths; that is, are drawn from deaths induced by the government measures, *via* the combined poverty, obesity and climatic factors, made potent by sustained chronic psychological stress, and from the deaths resulting from the direct assault against the elderly in March-June 2020 (cvp1) (Rancourt, 2020).

Let us examine these relations further. Figure 34c shows the P, I, C and PIC by week CDC data with our ACM-SB/w, 2014-2021, while Figure 34d shows the same data for the period 2019-2021.

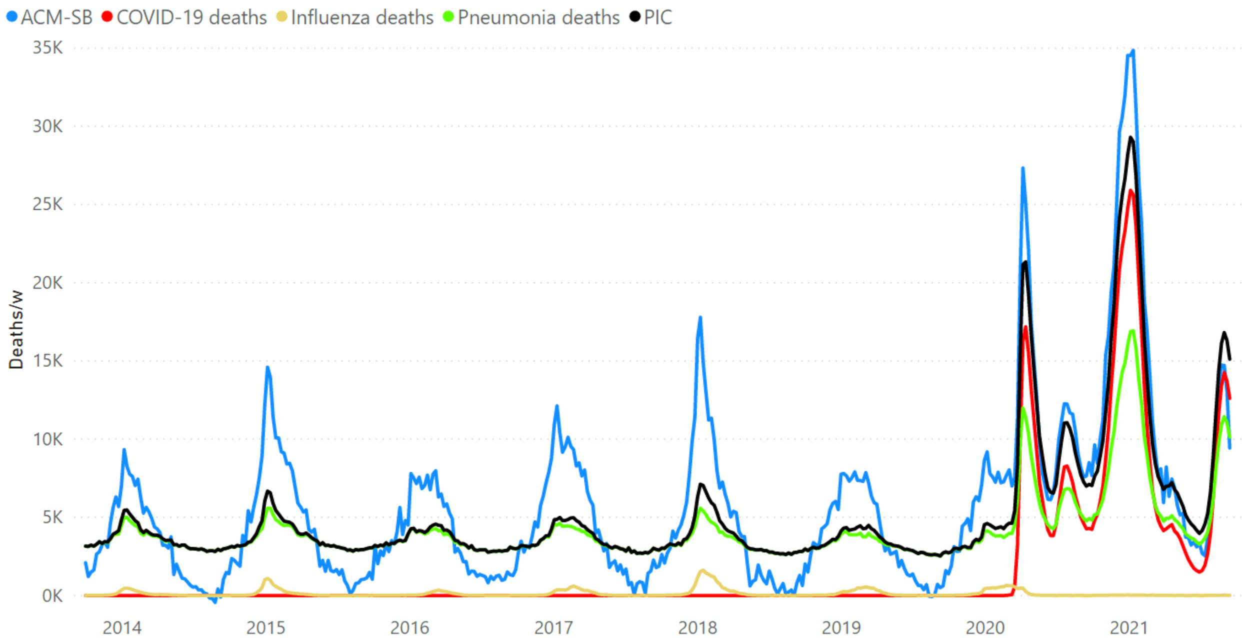


Figure 34c. All-cause above-SB (ACM-SB) (blue), COVID-19 (red), influenza (yellow), pneumonia (green) and PIC (black) mortality by week for the USA from 2014 to 2021. Data are displayed from week-40 of 2013 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. PIC is the deaths assigned to pneumonia and/or influenza and/or COVID-19. ACM and cause-assigned deaths data were retrieved from CDC (CDC, 2021a) as described in Table 1. SB was estimated as described in section 2.

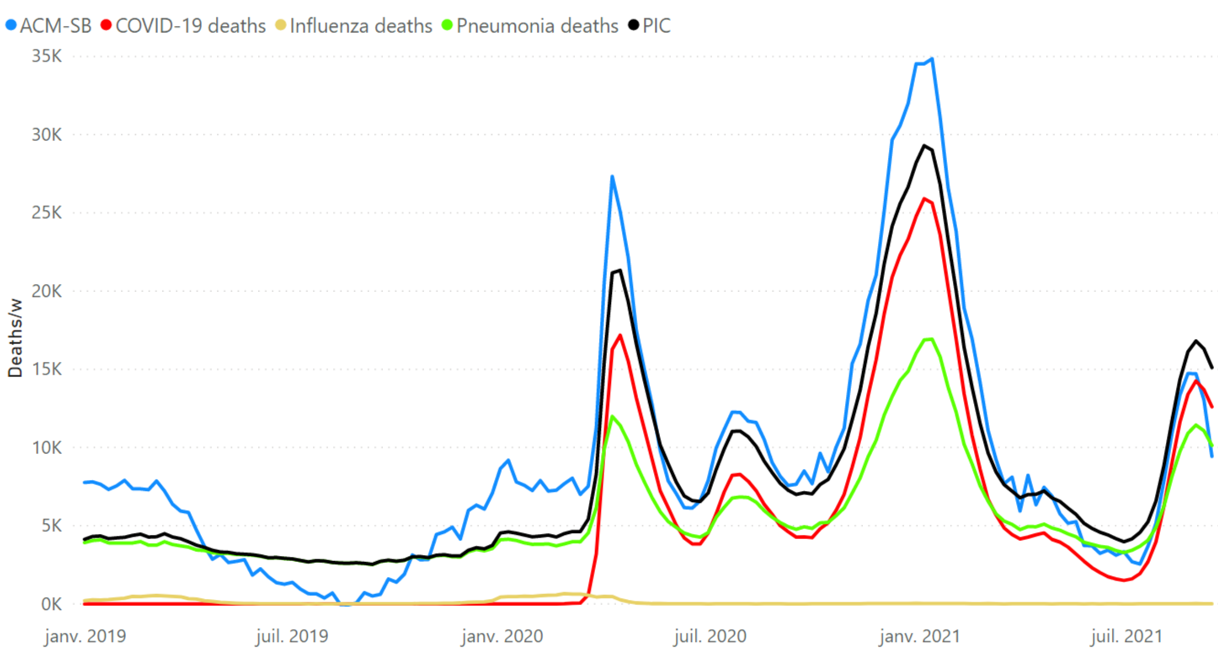


Figure 34d. All-cause above-SB (ACM-SB) (blue), COVID-19 (red), influenza (yellow), pneumonia (green) and PIC (black) mortality by week for the USA from 2019 to 2021. Data

are displayed from week-1 of 2019 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. PIC is the deaths assigned to pneumonia and/or influenza and/or COVID-19. ACM and cause-assigned deaths data were retrieved from CDC (CDC, 2021a) as described in Table 1. SB was estimated as described in section 2.

We note (Figures 34c and 34d) that pneumonia contributes significantly to summer deaths and that its summer-trough values are on a linear trend that is essentially horizontal for the years shown (approximately 2,680 pneumonia deaths per week, baseline). The same is true for PIC. Next, we therefore remove the “pneumonia-SB” (“pSB”) from the pneumonia data, and from the PIC data, in order to visualize solely deaths above summer-normal mortality.

The result is shown in Figure 34e (2014-2021) and Figure 34f (2019-2021).

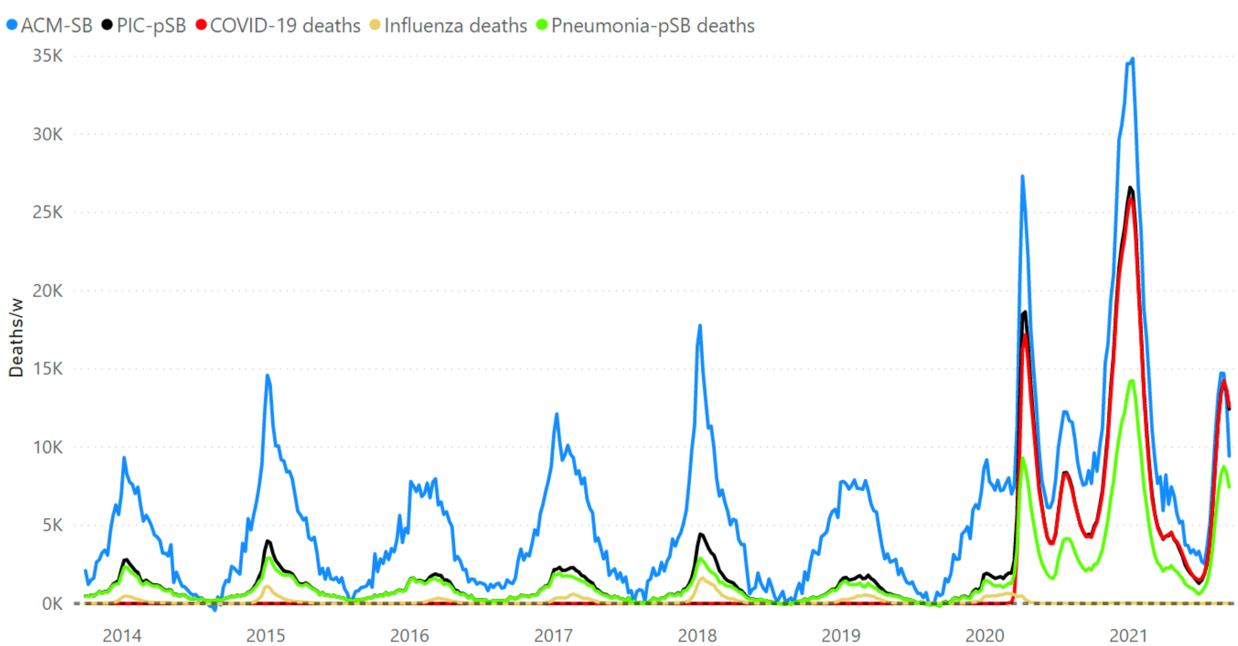


Figure 34e. All-cause above-SB (ACM-SB) (blue), COVID-19 (red), influenza (yellow), pneumonia-pSB (green) and PIC-pSB (black) mortality by week for the USA from 2014 to 2021. Data are displayed from week-40 of 2013 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The dashed line emphasizes the zero. pSB, the summer-trough pneumonia mortality, is removed from each week of pneumonia, and of PIC deaths. PIC is the deaths assigned to pneumonia and/or influenza and/or COVID-19. ACM and cause-assigned deaths data were retrieved from CDC (CDC, 2021a) as described in Table 1. SB was estimated as described in section 2.

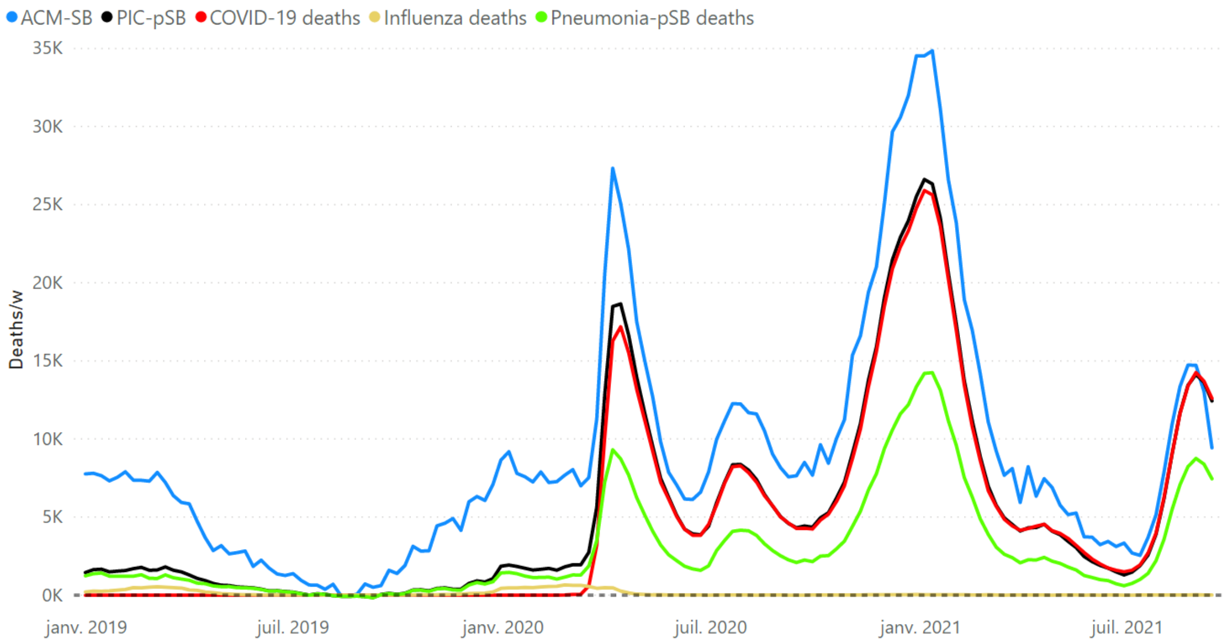


Figure 34f. All-cause above-SB (ACM-SB) (blue), COVID-19 (red), influenza (yellow), pneumonia-pSB (green) and PIC-pSB (black) mortality by week for the USA from 2019 to 2021. Data are displayed from week-1 of 2019 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The dashed line emphasizes the zero. pSB, the summer-trough pneumonia mortality, is removed from each week of pneumonia, and of PIC deaths. PIC is the deaths assigned to pneumonia and/or influenza and/or COVID-19. ACM and cause-assigned deaths data were retrieved from CDC (CDC, 2021a) as described in Table 1. SB was estimated as described in section 2.

Figures 34g and 34h show some of the same data as above but also the difference (residual) “ACM-SB” minus “PIC-pSB”, by week (black curve), for the USA. This difference (ACM-SB minus PIC-pSB) shows deaths that are not assigned to a respiratory disease (viral or any pneumonia) as a contributing cause of death.

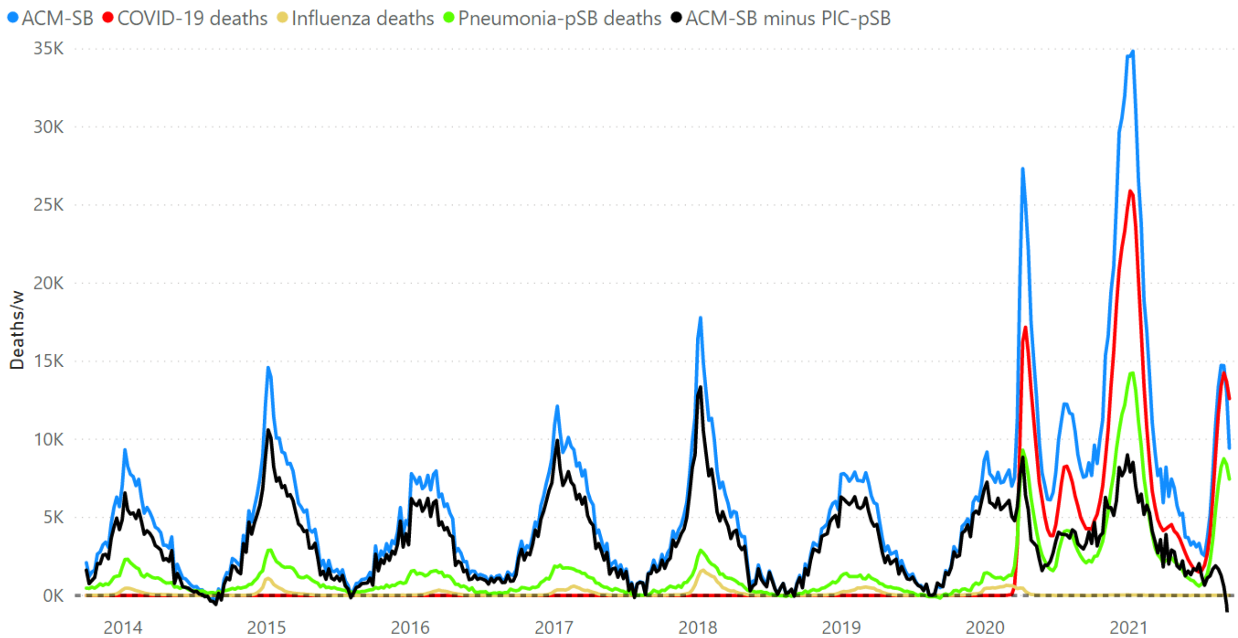


Figure 34g. All-cause above-SB (ACM-SB) (blue), COVID-19 (red), influenza (yellow), pneumonia-pSB (green) and ACM-SB minus PIC-pSB (black) mortality by week for the USA from 2014 to 2021. Data are displayed from week-40 of 2013 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The dashed line emphasizes the zero. pSB, the summer-trough pneumonia mortality, is removed from each week of pneumonia, and of PIC deaths. PIC is the deaths assigned to pneumonia and/or influenza and/or COVID-19. ACM and cause-assigned deaths data were retrieved from CDC (CDC, 2021a) as described in Table 1. SB was estimated as described in section 2.

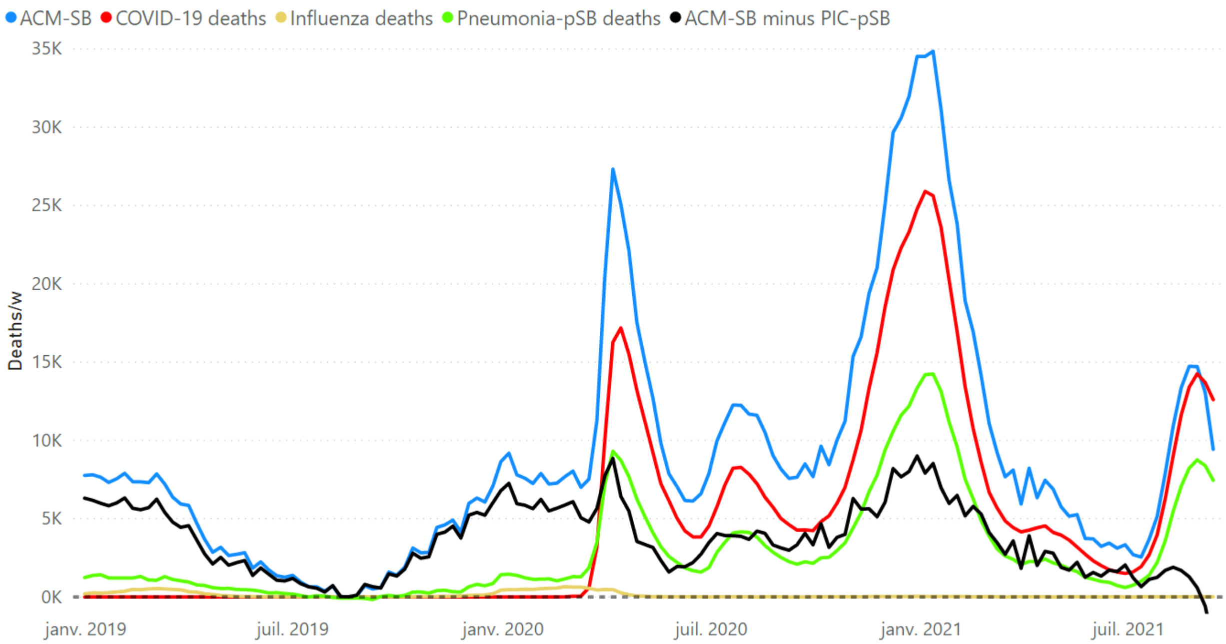


Figure 34h. All-cause above-SB (ACM-SB) (blue), COVID-19 (red), influenza (yellow), pneumonia-pSB (green) and ACM-SB minus PIC-pSB (black) mortality by week for the USA from 2019 to 2021. Data are displayed from week-1 of 2019 to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The dashed line emphasizes the zero. pSB, the summer-trough pneumonia mortality, is removed from each week of pneumonia, and of PIC deaths. PIC is the deaths assigned to pneumonia and/or influenza and/or COVID-19. ACM and cause-assigned deaths data were retrieved from CDC (CDC, 2021a) as described in Table 1. SB was estimated as described in section 2.

Figures 34a through 34h show that, in addition to COVID-19-associated deaths, there was a massive increase in pneumonia-associated deaths in the COVID-era in the USA, which had the same temporal pattern as both ACM and COVID-19-assigned deaths.

Figure 34i shows that COVID-19-assigned deaths were consistently associated with pneumonia as a contributing cause of death, some 40 to 60 % of the cases, throughout the COVID-era. Also, virtually all the above-pSB pneumonia assignations had COVID-19 co-assignations. That is, in number, all the excess pneumonia assignations in the COVID-era had COVID-19 co-assignations.

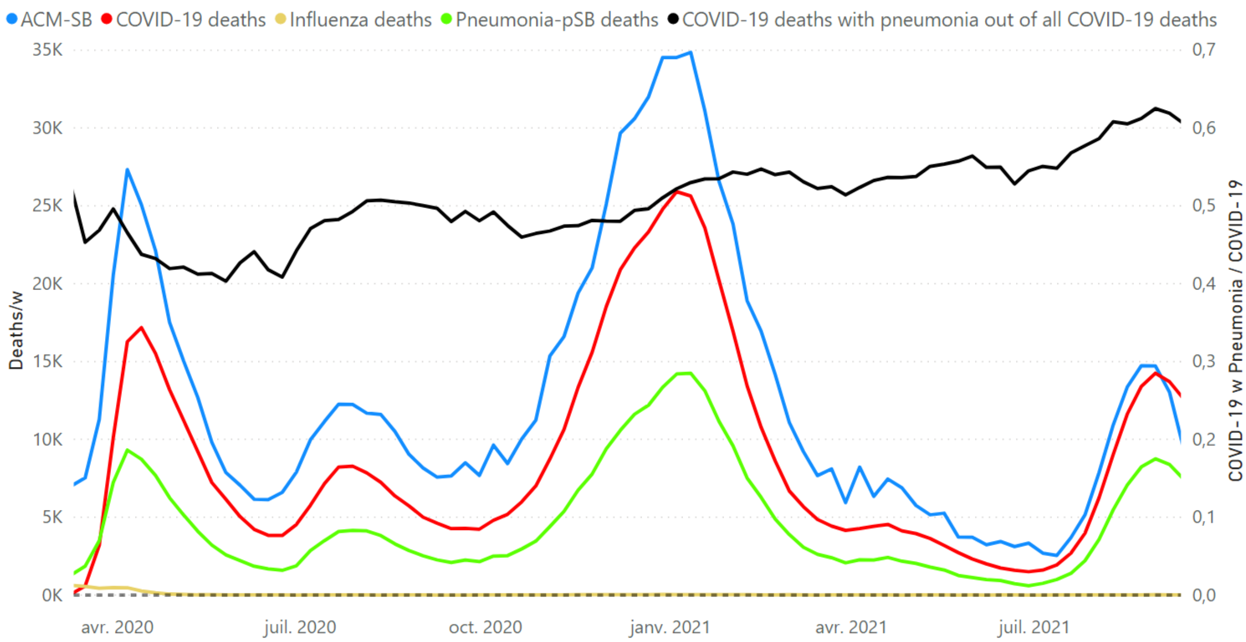


Figure 34i. All-cause above-SB (ACM-SB) (blue), COVID-19 (red), influenza (yellow) and pneumonia-pSB (green) mortality by week, and the ratio of COVID-19 deaths with pneumonia to all COVID-19 deaths (black, right Y-scale) by week, for the USA in the COVID-era (March-2020 into 2021). Data are displayed from week-11 of 2020 (week of March 11 2020, date of the WHO pronouncement of the pandemic) to week-37 of 2021 for the whole continental USA, including Alaska and Hawaii. The dashed line emphasizes the zero. pSB, the summer-trough pneumonia mortality, is removed from each week of pneumonia deaths. ACM and cause-assigned deaths data were retrieved from CDC (CDC, 2021a) as described in Table 1. SB was estimated as described in section 2.

The difference (ACM-SB minus PIC-pSB) shown in Figures 34g and 34h shows that excess (above-SB) deaths not assigned to a respiratory disease (viral or any pneumonia) as a contributing cause of death are approximately the same in number during the COVID-era as in previous years. Known causes of death for excess (above-SB, winter burden) deaths include heart disease, Alzheimer disease/dementia, and diabetes (Woolf et al., 2021). However, the difference (ACM-SB minus PIC-pSB) does show anomalies in the COVID-era: a sharp peak in March-May 2020, and a consistently large value in the summer-2020 period. A striking feature is that, unlike summer-2020, the rise in ACM-SB in summer-2021 is entirely assigned as PIC, virtually without any non-respiratory assignation.

The result that there were essentially no excess deaths (in number) assigned to non-respiratory causes in the COVID-era in the USA (Figure 34g) is surprising in that, for England and Wales, Kontopantelis et al. (2021) found, looking at excess deaths above historical trends, that in the first 30 weeks of the declared pandemic there were 62,321 excess deaths: 46,221 (74 %) attributable to respiratory causes, and 16,100 (26 %) to other causes.

Some authors have argued that COVID-19 deaths may be vastly underestimated by failing to correctly assign respiratory deaths to COVID-19 (Stokes et al., 2021) (IHME, 2021). We find this highly implausible for the USA. Acknowledging similar numbers of non-respiratory excess (above-SB) deaths in the COVID-era as in the pre-COVID-era (Figure 34g), leads one to conclude that virtually all other excess (above-SB) deaths (in number) in the COVID-era have been assigned as COVID-19, consistently including pneumonia as a jointly assigned cause of death in approximately 40-60 % of the thus COVID-19-assigned cases (Figure 34i). There is no room for more COVID-19 deaths in the USA accounting of mortality. Indeed, how could COVID-19-assignations be undercounted in the middle of the most mediatized, tested and medical-protocol regulated declared pandemic in memory, in a country that has some of the best medical statistics gathering in the world?

Respiratory causes appear to have been the main agent of death, regarding excess (above-SB) deaths in the USA in the COVID-era; however COVID-19 assignment remains suspect (Borger et al., 2021).

Shockingly, there was a massive epidemic or co-epidemic of pneumonia in the USA in the COVID-era, according to CDC data (CDC, 2021a) (Figure 34), which is never mentioned in the media and essentially not on the radar in the medical research literature. To the extent that there is COVID-19 over-assignation, it may represent up to 100 % of the COVID-era excess deaths from respiratory causes. It would not be the first time that the actual cause of a large epidemic is bacterial infection rather than the

presumed viral pathogen (Morens et al., 2008) (Chien et al., 2009) (Sheng et al., 2011). In the words of Ginsburg and Klugman (2020):

> Data regarding bacterial superinfections in COVID-19 pneumonia are still emerging, but an association has been made between the detection of bacterial products in blood with disease severity in COVID-19 patients.[ref] Diagnosing coinfections is complex in the best of circumstances and because there is a desire to avoid diagnostic procedures and minimise the exposure of COVID-19 to health-care workers, diagnosing potential bacterial superinfections during COVID-19 has been challenging.
> […] Although many serum biomarkers lack specificity, increased procalcitonin concentrations have been investigated as a specific bacterial differentiation from viral response to bacterial respiratory tract infection.[refs] From accumulating data and reports, there appears to be a clear association between elevated concentrations of procalcitonin and increasing COVID-19 disease severity, despite a variety of cutoffs chosen.[refs]
> Most bacterial pneumonias caught early enough can be safely and effectively treated with antibiotics […]

Vaccination

It is important to examine whether the large COVID vaccination campaign has had any influence on mortality and on the phenomena that we describe in this article. Figure 31 shows all-cause mortality by week (ACM/w), the number of total (all manufacturers) administered vaccines (doses/day) and the number of fully vaccinated individuals (vaccinated/day), on the same time axis, in the COVID-era (CDC, 2021a; CDC, 2021f).

An individual is considered fully vaccinated when second dose of a two-dose vaccine or one dose of a single-dose vaccine is completed (CDC, 2021f).

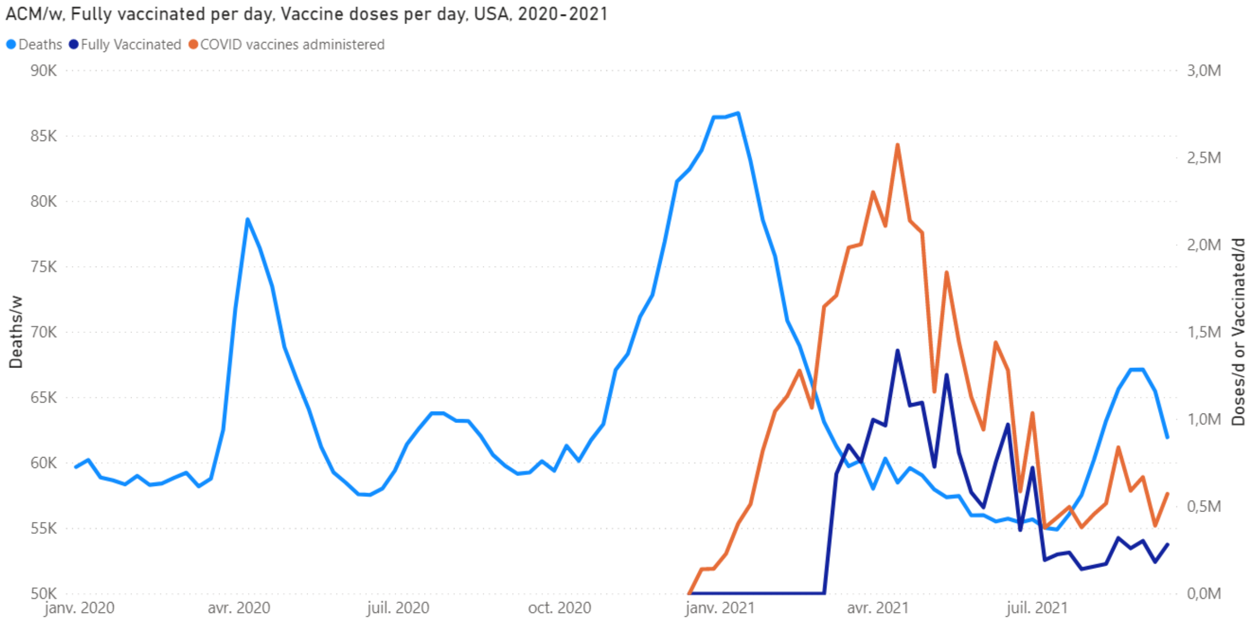


Figure 31. All-cause mortality by week (light blue), fully vaccinated individuals by day (dark blue) and COVID vaccine doses administered by day (orange), in the USA, from 2020 to 2021. Data are displayed from week-1 of 2020 to week-37 of 2021. For data by day, only one day a week is represented on the graph (Monday). An individual is considered fully vaccinated when second dose of a two-dose vaccine or one dose of a single-dose vaccine is completed. USA means 49 continental states, including the District of Columbia and excluding Alaska and Hawaii. Data were retrieved from CDC (CDC 2021a, CDC 2021f), as described in Table 1.

The total number of doses in the period illustrated is approximately 380 M and the total number of people being fully vaccinated is approximately 178 M. Therefore, the large hump in vaccinations per day constitutes the majority of the planned vaccination campaign (Figure 31).

Here (Figure 31), we note that our interpretations concerning cvp1 and smp1 mortality cannot be impacted whatsoever by vaccination because the vaccination injections and the fully vaccinated status started later, beyond the week of the inflection point on the rise of the cvp2 feature and towards the end of the cvp2 feature, respectively.

Readers who would be tempted to ascribe the downturn in the cvp2 peak to the vaccination campaign should note that the downturn coincides with the expected

seasonal downturn of every seasonal winter maximum that has ever been observed by epidemiologists in the last century or more.

More importantly, the largely completed vaccination campaign did not prevent a second surge of summer deaths (2021, “smp2”) (Figure 31). The mortality in the said second surge appears to be comparable to or more than the mortality for summer-2020. Furthermore, the COVID-19-assigned deaths (CDC, 2021a) are significantly greater in number in summer-2021 than in summer-2020 (Figure 34), and, unlike at any other time in the COVID-era, account for virtually all the excess (above-SB) deaths, in the summer-2021 feature (smp2) (Figure 34), following the vaccination campaign.

There is no sign in the ACM/w that the vaccination campaign has had any positive effect. However, given that the vaccination campaign starts well after the 2020 summer and essentially ends mid-summer-2021 prior to the start of the smp2 feature, given that the 2021 excess (above-SB) summer deaths (smp2) occur in significantly younger individuals than the excess summer-2020 deaths, and given that the smp2 feature is significantly larger than the smp1 feature for the said younger individuals (35-54 years, Figures 33d and 33e; and 55-64 years, Figure 33f, to a lesser degree), it is possible that vaccination made 35-54 year olds and others more vulnerable to death, especially summer death in disadvantaged individuals in hot-climate states (Montgomery et al., 2021) (Simone et al., 2021).

4. Comparison with Canada, and implications

One of the most striking aspects about mortality in the USA is that total yearly mortality in Canada is completely normal in the COVID-era: it lies precisely on the decadal trend established since 2010. We elaborated this fact about Canada in our recent article (Rancourt et al., 2021). At the time of publication, there was only enough weekly data to complete cycle-year 2020 for Canada. More data is now available, such that we can

now obtain cycle-year 2021, by implementing a short (10-week) reliable extrapolation to complete the needed summer-2021 trough section.

The latest Canadian data is shown in Figure 35.

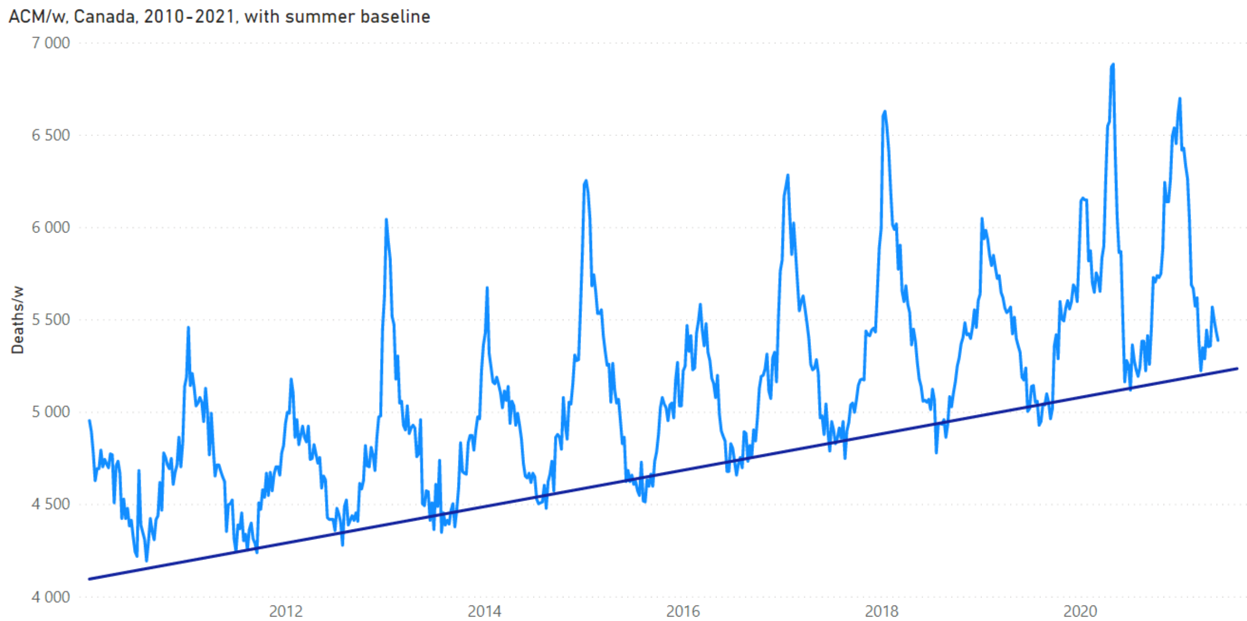


Figure 35. All-cause mortality by week in Canada from 2010 to 2021. The linear summer baseline (SB) is a least-squares fit to the summer troughs for summer-2013 through summer-2019, using the following summer trough weeks: 2013-weeks [24-37], 2014-weeks [28-33], 2015-weeks [27-37], 2016-weeks [24-34], 2017-weeks [25-34], 2018-weeks [28-35], 2019-weeks [26-38]. Data are displayed from week-1 of 2010 (week ending on January 9, 2010) to week-20 of 2021 (week ending on May 22, 2021) for the ACM and to week-30 of 2021 (week ending on July 31, 2021) for the SB. That way, the SB extends to the end of the 2021 cycle-year (week-30 of 2021), thereby showing the segment needing extrapolation discussed in the text. Data were retrieved from StatCan (StatCan, 2021), as described in Table 1.

The said extrapolation is performed as follows. We work with ACM-SB/w, average the values for 2021 weeks 10 through 20, which is a relatively flat region in ACM-SB/w, in the summer 2021 "trough" (week 20 is the last usable week in the data), and this average value is adopted for weeks 21 through 30 in ACM-SB/w (week 30 is the last week of the 2021 cycle-year). We then take this ACM-SB/w (including the thus extrapolated 10-week segment) and transform back to an ACM/w by adding the SB. The total mortalities per cycle-year are then calculated from sums on this ACM/w data,

which now is extended to complete the last (2021) cycle-year. The extrapolation is an accurate representation of the last 10 weeks in the 2021 cycle-year, unless something unexpected and significant occurs in those 10 weeks in mid-summer-2021, beyond the already higher summer-trough values occurring in the COVID-era for Canada (Figure 35).

The resulting ACM per cycle-year versus cycle-year for Canada is shown in Figure 36, with a best-line fit to illustrate the trend.

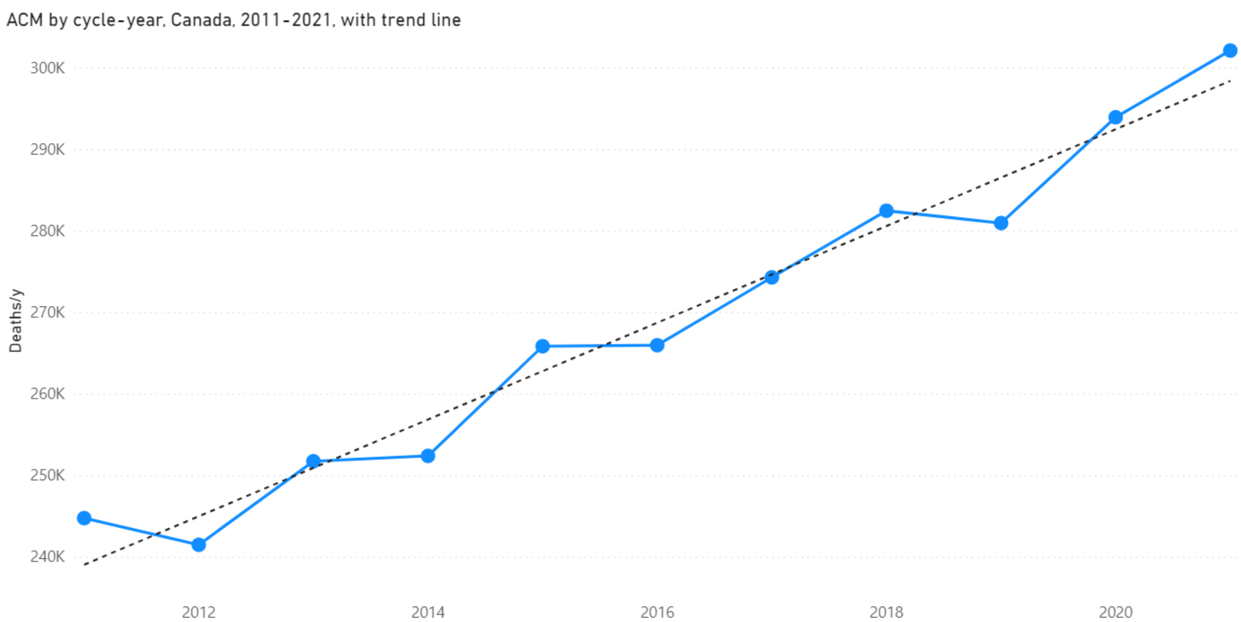


Figure 36a. All-cause mortality by cycle-year for Canada, cycle-years 2011 to 2021. The dashed line is a least-squares fitted straight line. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). The ACM over the weeks 21 to 30 of 2021 was extrapolated, as described in the text, in order to complete the 2021 cycle-year. Raw data were retrieved from StatCan (StatCan, 2021), as described in Table 1.

Figure 36a is the same as Figure 2 in our prior article (Rancourt et al., 2021), except for the addition of one more cycle-year (2021). This further confirms that “there was no pandemic in Canada” (Rancourt et al., 2021).

We also calculated the WB of deaths for cycle-years 2011 through 2021, which is shown in Figure 36b. A slight increase by year is expected because the population of those most vulnerable to winter-time deaths is increasing. Again, as with ACM itself, nothing in the values of WB deaths indicates any pandemic or any unusual additional cause of yearly mortality in cycle-years 2020 or 2021.

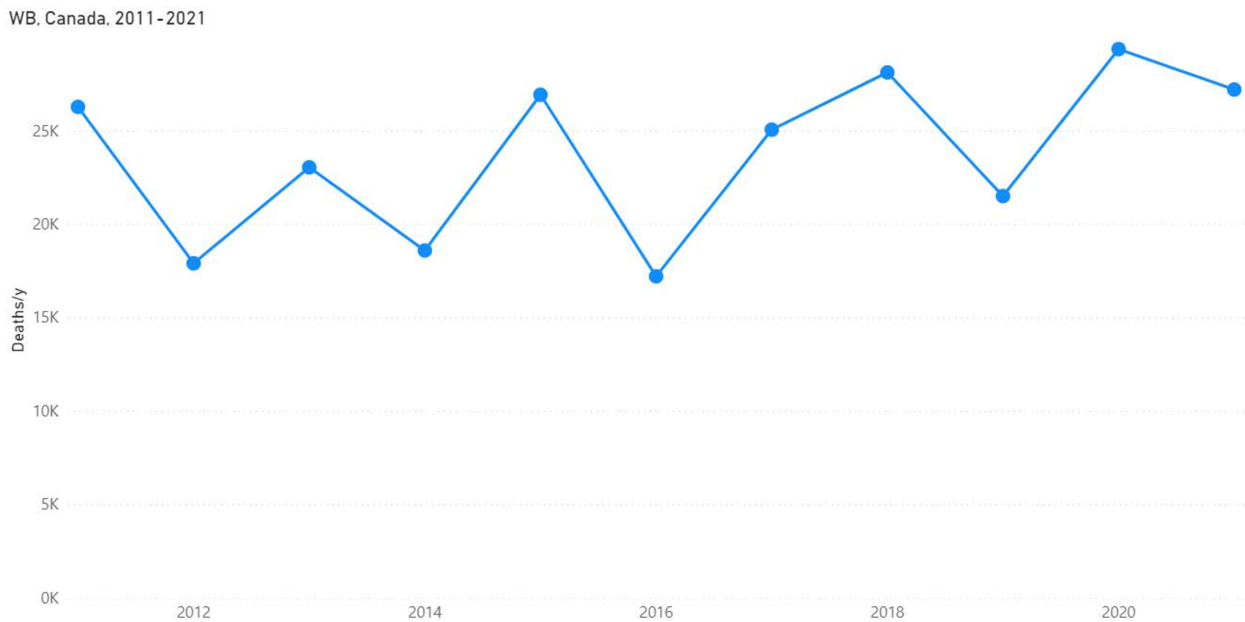


Figure 36b. Winter burden (WB) for Canada for cycle-years 2011 to 2021. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). The ACM-SB over the weeks 21 to 30 of 2021 was extrapolated, as described in the text, in order to complete the WB of the cycle-year 2021. Raw data were retrieved from StatCan (StatCan, 2021), as described in Table 1.

The ACM/w can also be used to calculate ACM by calendar-year, which is shown, compared to ACM by cycle-year, in Figure 37 for Canada.

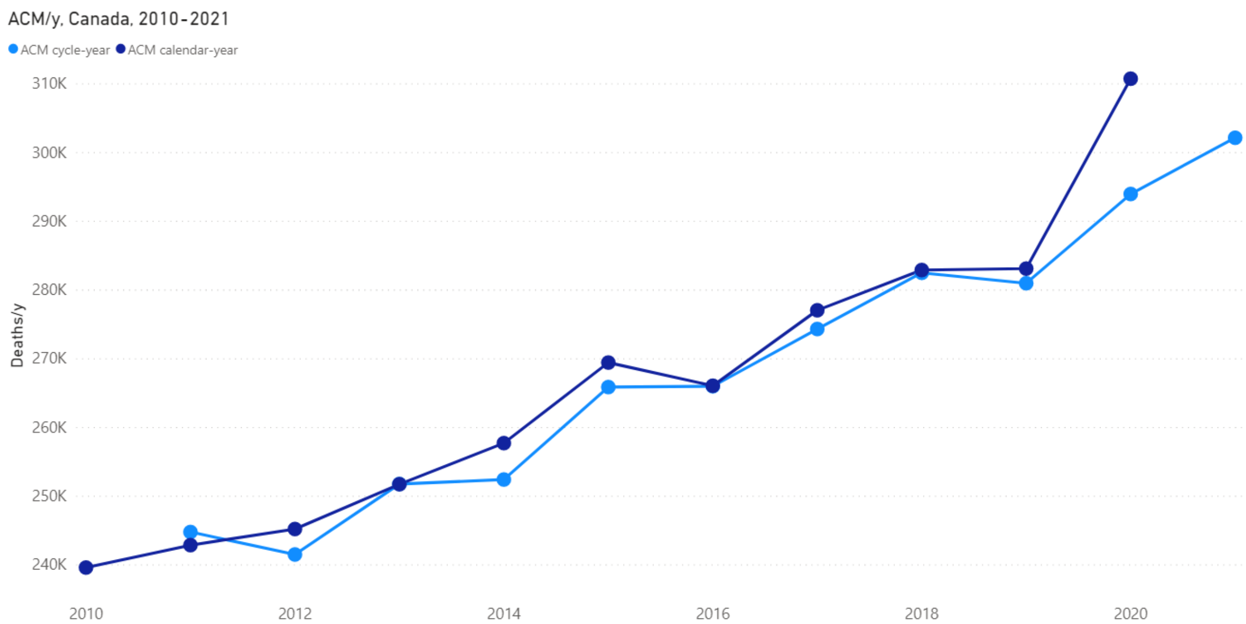


Figure 37. All-cause mortality by calendar-year (dark blue), calendar-years 2010 to 2020, shown with all-cause mortality by cycle-year (light blue), cycle-years 2011 to 2021, for Canada. Cycle-year N means the period from mid-summer of calendar-year N-1 to mid-summer of calendar-year N. The ACM over the weeks 21 to 30 of 2021 was extrapolated, as described in the text, in order to complete the 2021 cycle-year. Raw data were retrieved from StatCan (StatCan, 2021), as described in Table 1.

In Figure 37 the ACM by calendar-year for 2020 is higher than the visible trend because of an accident in the positions of ACM/w peaks: there is a large late peak in cycle-year 2020 (the March-June 2020 so-called “covid” peak, or “cvp1”) and a large early rise in the winter peak of cycle-year 2021. In this figure, recall that cycle-year N means the period from mid-summer of calendar-year N-1 to mid-summer of calendar-year N.

Clearly, there is no sign of a pandemic in Canada, or of a COVID-era anomaly, in terms purely of ACM by cycle-year and WB (Figure 36), which is at odds with the dramatic increase seen for the neighbouring USA: Figure 1, by calendar-year up to 2020; Figure 5, in the ACM/w data itself; Figure 12a, expressed as WB versus cycle-year.

If a new pathogen caused the havoc that we have described for the USA during the COVID-era, then how could such a virulent and contagious pathogen not have crossed the world's longest international land border (8,890 km) between two major trading

partners? Did Canada apply effective mitigation strategies, completely different from those applied in the major states of the USA, which reduced the mortality impact of the new pathogen to zero on the Canadian territory? The answers must be “that would be impossible” and “no”, respectively.

Viral respiratory diseases, in particular, are believed to be very contagious, and more so for presumed pandemic-causing new viruses for which there is no prior immunity in the world populations. Either the presumed new virus was not able to cross the USA-Canada border or Canadians of heterogeneous origins are genetically resilient to the new virus or the massive excess deaths in the USA during the COVID-era are not primarily due to any new respiratory virus. We think the latter must be concluded, and this is consistent with our findings of co-correlations with socio-geo-economic and climatic factors, which project to zero excess deaths for sufficiently small values of the correlated or co-correlated factors (e.g., Figure 25, for summer-2020 deaths).

5. Mechanistic causes for COVID-era deaths

To be clear, we have not shown that USA deaths are correlated to poverty, obesity and hot climatic regions, although that in itself is probably true to a significant degree, as can be inferred from a map of life expectancy at birth by state of the USA, such as the one shown in Figure 38a.

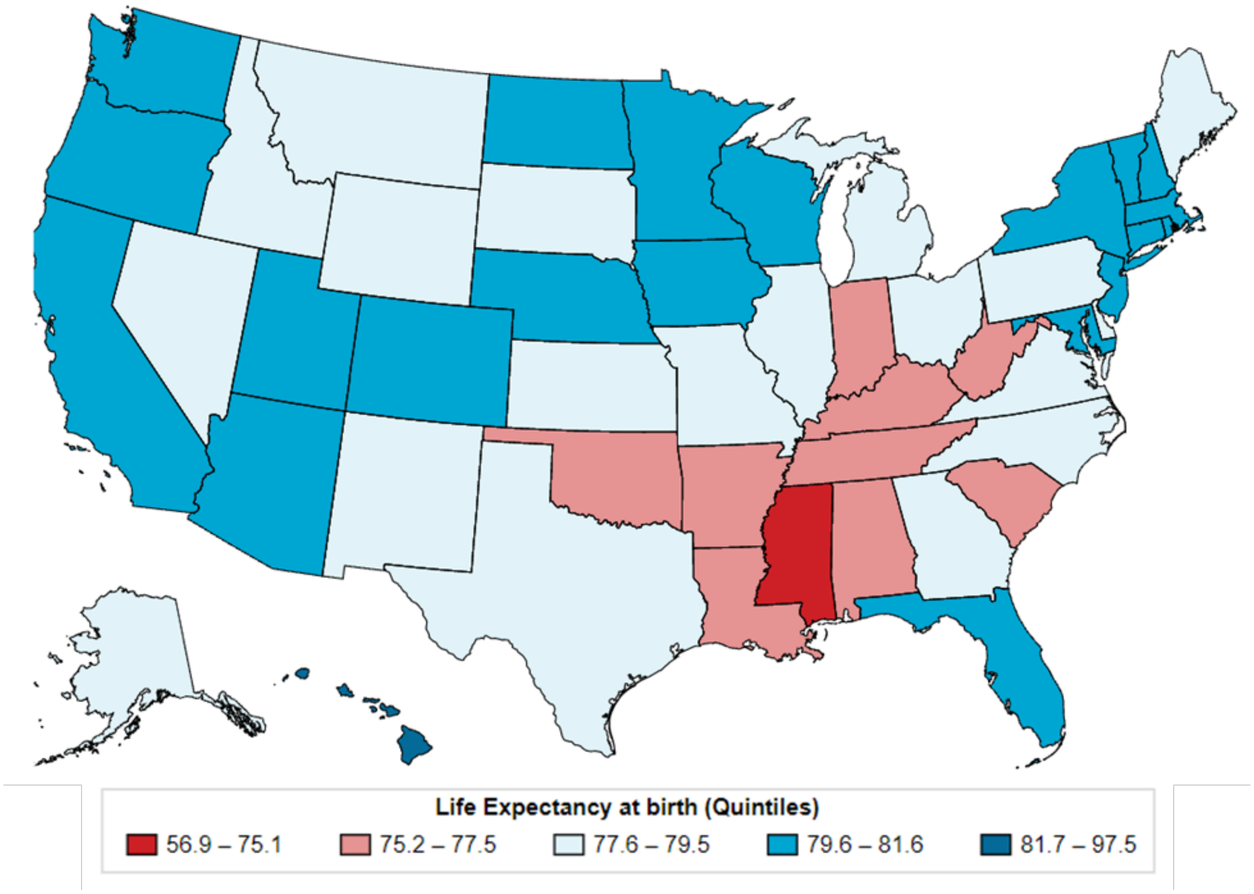


Figure 38a. Map of life expectancy at birth for USA states, from census tracts 2010-2015 (Tejada-Vera et al., 2020). Present interactive map location: https://www.cdc.gov/nchs/data-visualization/life-expectancy/index.html

This map of life expectancy at birth by state (Figure 38a) is in turn very similar to a map of antibiotic prescriptions by population by state, such as the one shown in Figure 38b.

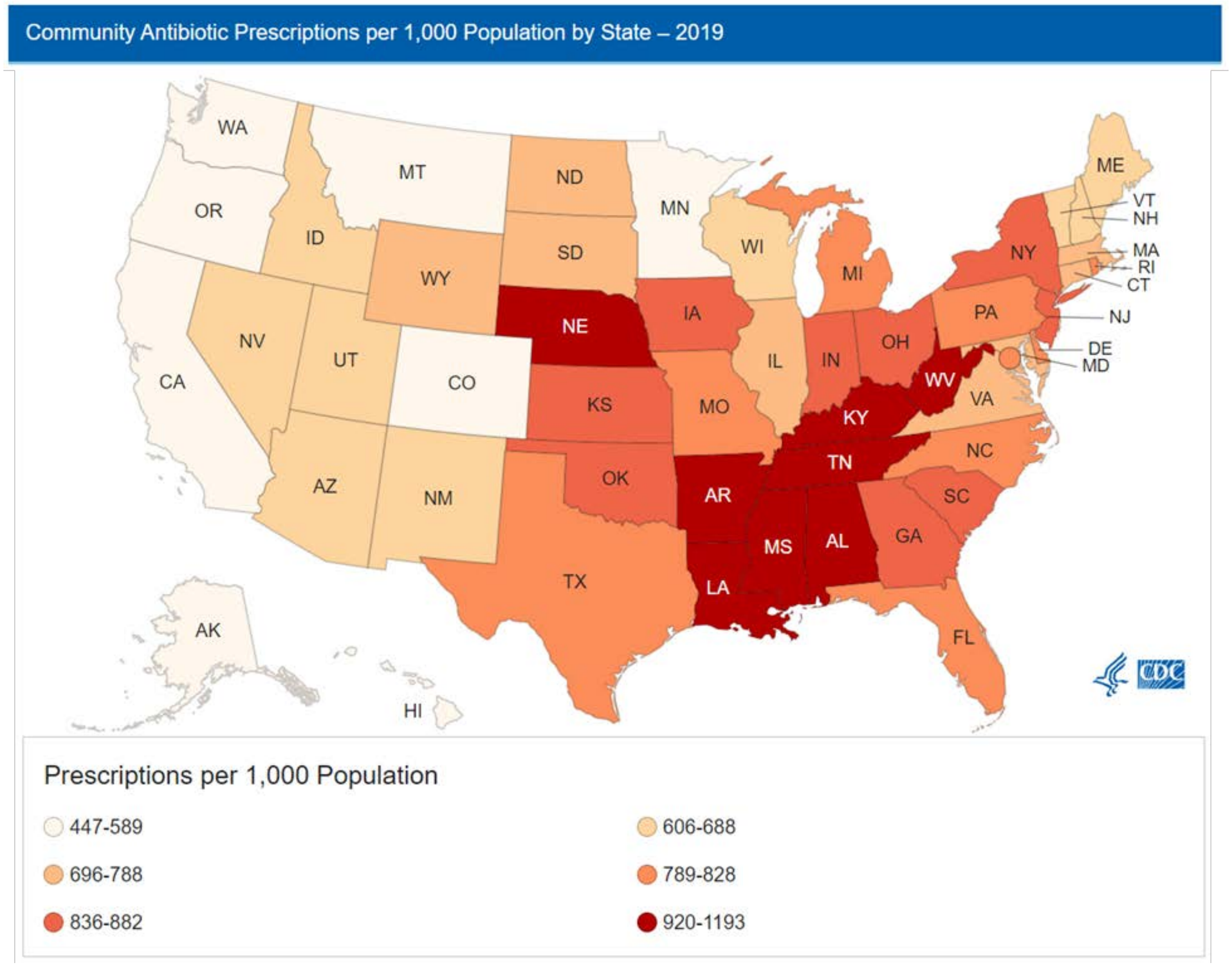


Figure 38b. Antibiotic prescriptions per 1,000 persons by state (sextiles) for all ages, United States, 2019. "Healthcare providers prescribed 251.1 million antibiotic prescriptions—equivalent to 765 antibiotic prescriptions per 1,000 persons", in 2019 (CDC, 2021g).

Given the similarity in state-wise distributions of life expectancy at birth (Figure 38a) and antibiotic prescriptions (Figure 38b), it is not unreasonable to conclude that a dominant cause of death limiting life expectancy, in the USA in the pre-COVID-era, is bacterial infection, the most common fatal such infection being bacterial pneumonia.

However, what we have shown is that, in the COVID-era, during summer-2020 (smp1), fall-winter-2020-2021 (cvp2) and summer-2021 (smp2), combined factors including poverty, obesity and hot climate became deadly associations for excess (above-SB)

deaths, beyond the deaths that would have occurred from the pre-COVID-era background of preexisting risk factors.

In addition, we have repeatedly concluded that the sharp peak in excess mortality occurring in March-June 2020 in some USA states (“covid” peak) (cvp1) must be a consequence of aggressive government and medical response to the WHO 11 March 2020 declaration of a pandemic, in those hot-spot jurisdictions, such as New York City in particular in the USA, and we have outlined likely mechanisms whereby this aggression would have caused a large surge of deaths in care homes and hospitals everywhere that it occurred (Rancourt, 2020) (Rancourt et al., 2020) (Rancourt et al., 2021).

The question now arises: By what mechanism(s) did the COVID-era government and medical disruptions induce excess deaths, at the population level, in the most vulnerable populations (elderly, and poverty + obesity + hot climate)? Alternatively (Figure 34), by what mechanism(s) did the COVID-era government and medical disruptions make respiratory diseases, including pneumonia, so much more fatal than usual, at the population level, in the most vulnerable populations (elderly, and poverty + obesity + hot climate)? What about the COVID-era so dramatically multiplied the deadliness of poverty + obesity + hot climate, in the USA?

We submit that the overly succinct three-word answer is: “chronic psychological stress”, plus deadly institutional aggression and neglect of the sick elderly regarding the March-June 2020 catastrophe (cvp1). “Chronic psychological stress” is a powerful determinant of individual health (see below), which is essentially ignored by all those who accept the promoted dominant view that the virulence and contagiousness of the viral respiratory pathogens are predominantly determined by viral genetics, with only secondary influence from host characteristics and social determinants of host characteristics. The dominant view is contradicted by more than a century of hard mortality data, as explained above (Figures 1 through 4), where the declared pandemics are undetected

and all the detected major mortality excesses are tied to socio-economic periods and events.

Researchers considering mortality from diseases must make themselves aware that ordinary psychological stress significantly impacts immune response, and that psychoneuroimmunology is a large field of research (Ader and Cohen, 1993).

Social status, within a specific dominance hierarchy, is a major predictor of chronic stress, in social animals including humans (Cohen et al., 1997a) (Sapolsky, 2005), which, in turn, may be the dominant determinant of individual health, disease burden, and longevity (Cohen et al., 2007).

Ordinary psychological stress is known to be a dominant factor in making an individual susceptible to viral respiratory disease symptomatic infection, and to increase the severity of the infection (Cohen et al., 1991). Also, social isolation (paucity of social-network interactions), in addition to individual psychological stress, is known to have an added impact on the individual's susceptibility to viral respiratory diseases (Cohen et al., 1997b).

Furthermore, there is a large age gradient for stress endurance: extended periods of psychological stress are known to have more deleterious health effects in elderly persons than in younger persons (Prenderville et al., 2015).

The stress-immune relationship, however, is not simply a monotonic function of integrated intensity. Frequency and duration are pivotal: chronic or long-term stress harms immune response, whereas short-term adaptive stress enhances immune response. The often-cited review by Dhabhar (2014) has:

> Short-term (i.e., lasting for minutes to hours) stress experienced during immune activation enhances innate/primary and adaptive/secondary immune responses. Mechanisms of immuno-enhancement include changes in dendritic cell, neutrophil,

> macrophage, and lymphocyte trafficking, maturation, and function as well as local and systemic production of cytokines. In contrast, long-term stress suppresses or dysregulates innate and adaptive immune responses by altering the Type 1–Type 2 cytokine balance, inducing low-grade chronic inflammation, and suppressing numbers, trafficking, and function of immunoprotective cells.

Peters et al*.* (2021) have reviewed these concepts and the known science for the relevance to COVID-19. They pointed out that "the socioeconomic issues and various aspects of the Western type lifestyle that are closely associated with psychosocial stress have recently been reported to contribute to COVID-19". Their ultimate aim is to "clarify whether psychosocial interventions have the potential to optimize neuroendocrine-immune responses against respiratory viral infections during and beyond the COVID-19 pandemic."

Therefore, it is not difficult to imagine that the massive socio-economic disruptions of the COVID-era would have caused undue chronic psychological stress and amplified dominance-hierarchy stress predominantly against those who are already at the bottom of the societal dominance hierarchy, and have the least means to adjust to dramatically new circumstances. The new circumstances include: loss of sources of income, both legitimate and illegal, increased social isolation, increased hierarchical impositions, constant fear propaganda, severe mobility restrictions, closing of public and corporate-public spaces previously used, enforcement and intimidation against private or informal gatherings, mobbing against those who do not cheerfully accept the "new reality", and increased aggressions from equally stressed individuals. The missing means to adjust would include: undisturbed salary and ability to work from home, means to stay connected by Zoom (by video conferencing applications), large comfortable air-conditioned homes, means to home-school children in an adapted environment, nearby facilities for outside exercise, private facilities for physical exercise, undisturbed shopping by home delivery, undisturbed self-medication, continued access to health care, and so on.

It follows, from the science reviewed above, that the “undue chronic psychological stress and amplified dominance-hierarchy stress”, generally applied to entire populations, would cause death in those most likely to experience the stress and already in higher risk categories. It appears, for example, that populations normally adapted to summer heatwaves in the Southern USA were either prevented from practicing their usual adaptations to the heat or became more vulnerable to this physiological stress, or both.

It is evident also that the type of weakening of the immune system caused by chronic psychological stress would lessen the body’s ability to fight bacterial pneumonia, and that the populations hardest hit during the COVID-era are already disproportionately susceptible to bacterial pneumonia (Figure 38).

At this stage (Figure 34, Figure 38), and given the state of science and practice in this regard (Ginsburg and Klugman, 2020), it is not unreasonable to ask whether the logic has not been inverted: Is COVID-19-assignment an incorrect cause-assignment for what is in fact bacterial pneumonia? From this perspective, it becomes relevant to point out that Ivermectin is probably an effective antibacterial agent against tuberculosis, for example (Crump, 2017) (Lim et al., 2013), which would have been prescribed where the mainstream protocols call for avoiding antibiotics (Beovic et al., 2020) (CDC, 2021h) (Karami et al., 2021).

Karami et al. (2021) put it this way:

> Conclusions: On presentation to the hospital bacterial co-infections are rare, while empiric antibiotic use is abundant. This implies that in patients with COVID-19 empiric antibiotic should be withheld. This has the potential to dramatically reduce the current overuse of antibiotics in the COVID-19 pandemic.

Buehrle et al. (2020) pointed out that, at the same time, outpatient antibiotic prescriptions dropped significantly in the USA:

> Abstract: In April 2020, there were significant reductions in prescription fills of each of the 10 most prescribed outpatient antibiotics in the United States. Monthly azithromycin, amoxicillin-clavulanate, and levofloxacin fills did not rebound significantly from April through July 2020. Coronavirus disease 2019 had an immediate and sustained impact on US outpatient antibiotic prescribing.

The CDC (2021h) shows this graph:

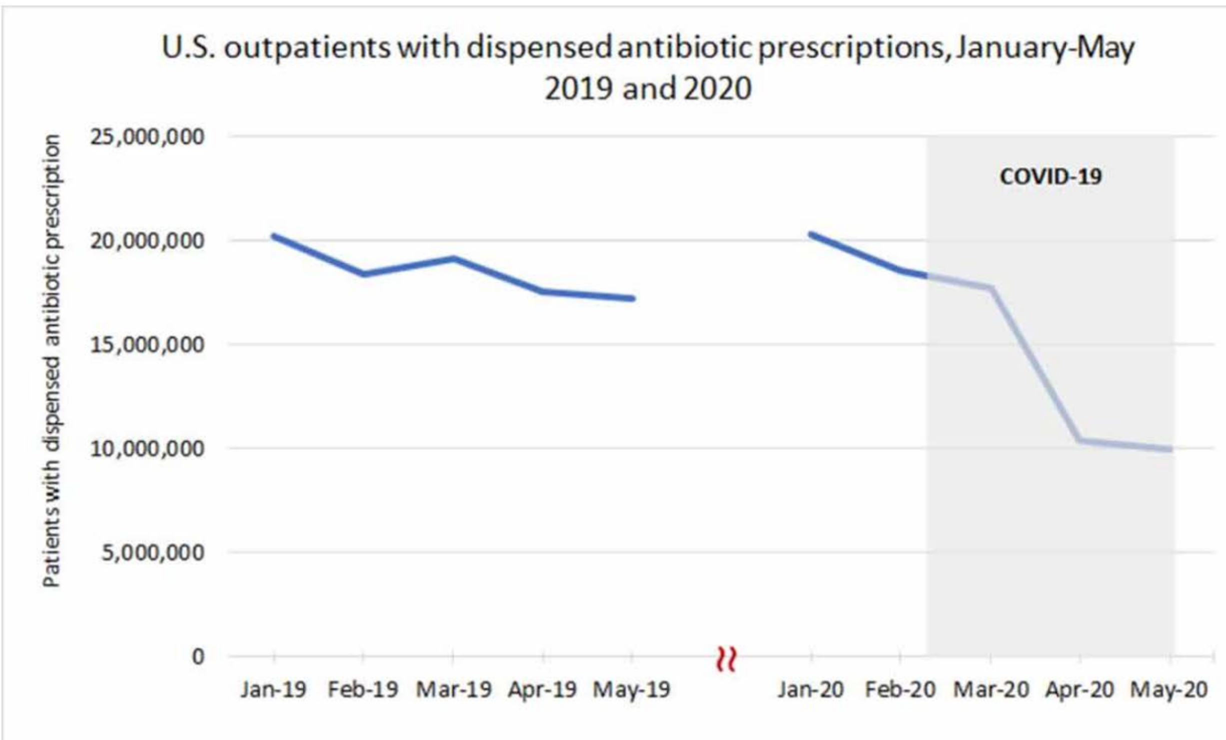


Figure 39. Estimated number of outpatients with dispensed antibiotic prescriptions, USA, 2019-2020. (CDC, 2021h).

If COVID-19 is largely misdiagnosed bacterial pneumonia (using a faulty PCR test: Borger et al., 2021; or not using any laboratory test), or if co-infection with bacterial pneumonia is not appropriately recognized (Ginsburg and Klugman, 2020), or if bacterial pneumonia itself goes otherwise untreated, while antibiotics (and Ivermectin) are withdrawn, in circumstances where large populations of vulnerable and susceptible residents have suppressed immune systems from chronic psychological stress induced

by large-scale socio-economic disruption, then the state has recreated the conditions that produced the horrendous bacterial pneumonia epidemic of 1918 (Morens et al., 2008) (Chien et al., 2009) (Sheng et al., 2011), in COVID-era USA.

6. Conclusion

By examining the socio-jurisdictional and temporal structure of the ACM/w data, and by comparing to socio-geo-economic and climatic data, we conclude that the massive above-trend COVID-era mortality in the USA is not the result of a pandemic, but instead is caused by the large-scale medical and government responses, which transformed the domestic economy and living conditions, and the associated long-term chronic psychological stress effects on the most vulnerable populations (regarding poverty and obesity), in a context of ordinary seasonal respiratory diseases and typical summer heat-wave climatic effects.

In light of the results presented herein, the view that a new respiratory disease virus caused the excess deaths in the COVID-era (March-2020 to present) in the USA has to be considered an extravagant theory, contrary to empirical data and viral respiratory disease phenomenology:

- No declared pandemic (1957-58, 1968, 2009) has ever caused a detectable increase in yearly all-cause mortality in the USA, since 1900, except 1918, which has been incorrectly assigned as an influenza pandemic.
- All the detected anomalies in yearly all-cause mortality in the USA, since 1900, have been associated with major socio-economic upheavals: the First World War, The Great Depression and Dust Bowl, the Second World War, and the medical and government response to the declared COVID-19 pandemic.
- None of the recently declared viral respiratory disease pandemics (1957-58, 1968, 2009), and none of the ubiquitous seasonal (winter) epidemics of the last

century or more, in all Northern hemisphere countries having sufficiently good data, exhibit large jurisdictional heterogeneity (in both time and location) in all-cause mortality of the magnitude seen during the COVID-era.

- On the contrary, viral respiratory disease epidemics, never mind declared pandemics, never stop at jurisdictional boundaries or national or state or provincial or regional or county borders. Instead, seasonal (winter) all-cause mortality is always synchronous across mid-latitude Northern hemispheric jurisdictions, while showing similar to statistically identical patterns of temporal variation within any given year.
- The jurisdictional and temporal heterogeneity of all-cause mortality during the COVID-era in the USA (and other nations) is of unprecedented character and magnitude (Figures 5-11, 13-16, and Table 2), which can only be due to local and time-dependent forces and vulnerability to those forces, not viral respiratory diseases as the primary driver.
- The extraordinary mortality spike that occurred in New York City and some North-East coastal states in March-June 2020 (cvp1) and virtually nowhere else (some 34 USA states did not significantly exhibit this feature in all-cause mortality) is impossible for a virulent and contagious respiratory disease virus acting in a society free from local aggression or local environmental disaster. To our knowledge, no such intense feature, this late in the cycle-year, has ever occurred in the world epidemiological record.
- Viral respiratory diseases never give rise to all-cause mortality by time peaks (maxima) in the summer. The unprecedented summer peaks seen in the USA in the COVID-era are contrary to known viral respiratory disease epidemiology.
- Pre-COVID-era viral-respiratory-disease burden mortality (winter burden) does not correlate with obesity, whereas the state-wise heterogeneous summer-2020, fall-winter-2020-2021 and summer-2021 excess (above-SB) mortalities do correlate with obesity.
- Pre-COVID-era viral-respiratory-disease burden mortality (winter burden) does not correlate with poverty, whereas the state-wise heterogeneous summer-2020,

fall-winter-2020-2021 and summer-2021 excess (above-SB) mortalities do correlate with poverty.

- Pre-COVID-era viral-respiratory-disease burden mortality (winter burden) does not correlate with climatic temperature, whereas the state-wise heterogeneous summer-2020, fall-winter-2020-2021 and summer-2021 excess (above-SB) mortalities do correlate with climatic temperature.
- In the correlations that we identified, the 2020 and 2021 summer excess (above-SB) mortalities extend to zero values for sufficiently small values of poverty, obesity or summer temperatures, or their combinations, such as the product of poverty and obesity, suggesting that the presumed new pathogen requires sufficiently high state-wise average poverty, obesity and/or temperatures in order to spread and be lethal in the summer.
- Pre-COVID-era viral-respiratory-disease burden mortality (winter burden) always correlates with the proportion of the population that is elderly, whereas the state-wise heterogeneous summer-2020, fall-winter-2020-2021 and summer-2021 excess (above-SB) mortalities anti-correlate with the proportion of the population that is elderly, strongly so for summer mortality.
- No known respiratory disease virus has ever caused a permanent (1.5 years and counting) step-wise time-independent increase in mortality of 15-34 year olds, which appears to have occurred in the COVID-era (Figures 33b to 33e).
- Pre-COVID-era viral-respiratory-disease burden mortality (winter burden) does not correlate with population density (Figure 29), whereas the state-wise heterogeneous March-June 2020 excess mortality (cvp1) strongly correlates with population density; and summer-2020, fall-winter-2020-2021 and summer-2021 excess (above-SB) mortalities anti-correlate with population density (Figure 30). (This is a consequence of the localities of the March-June 2020 anomaly, and that poor states tend to have low population density.)
- The largest high-tech vaccination campaign in history, targeted against the presumed pathogen, had no detectable benefit in all-cause mortality, given the post-vaccination-campaign summer-2021 surge that is observed.

- It is extremely unlikely that a virulent and contagious viral respiratory pathogen that would have caused the exceedingly large COVID-era excess mortality in the USA, could not have crossed the border into Canada, the world's longest international land border (8,890 km) between two major trading partners; where both countries are normally (pre-COVID-era) continuously subject to seasonal (winter) viral respiratory disease epidemics having virtually identical mortality characteristics.

Finally, our examination of plausible mechanisms for the exceptionally large COVID-era mortality in the USA, given all our empirical observations, leads us to postulate that COVID-19 may largely be misdiagnosed bacterial pneumonia (using a faulty PCR test: Borger et al., 2021; and see Ginsburg and Klugman, 2020), that correctly assigned bacterial pneumonia itself largely goes untreated, while antibiotics (and Ivermectin) are withdrawn, in circumstances where large populations of vulnerable and susceptible residents have suppressed immune systems from chronic psychological stress induced by ("COVID response") large-scale socio-economic disruption, and that the USA has, in the COVID-era, thus recreated the conditions that produced the horrendous bacterial pneumonia epidemic of 1918 (Morens et al., 2008) (Chien et al., 2009) (Sheng et al., 2011).

Given the approximately 1 M excess deaths that have occurred in the most vulnerable and underprivileged residents of the USA in the COVID-era, given the evidence from empirical and statistical data on the causes of the excess mortality, and in view of our research and general observations, we feel justified in making the following comment. We believe that genetic-sequencing-centered virologists and mathematical modellers (as opposed to other and broad disciplines connected to epidemiology, biology, psychology and health), pharmaceutical-industry lobbyists, politicized public health officials (WHO, national, and local), biased media, and approval-seeking politicians, have had far too much influence on public policy in the events surrounding the proclaimed pandemic, and in establishing the questionable dominant narrative, without regard for the hard data that is all-cause mortality by time, jurisdiction, age group, sex,

and so forth; without regard for robust measures of population-level actual harm, while allowing tunnel-vision assignation of cause. The resulting practice has been mostly contrary to public health principles of objectively, scientifically, equally and independently assessing risks and benefits of any impactful policy, within a framework of transparency and accountability; and has caused great societal harm, beyond significant excess mortality itself, which is difficult to fully quantify.

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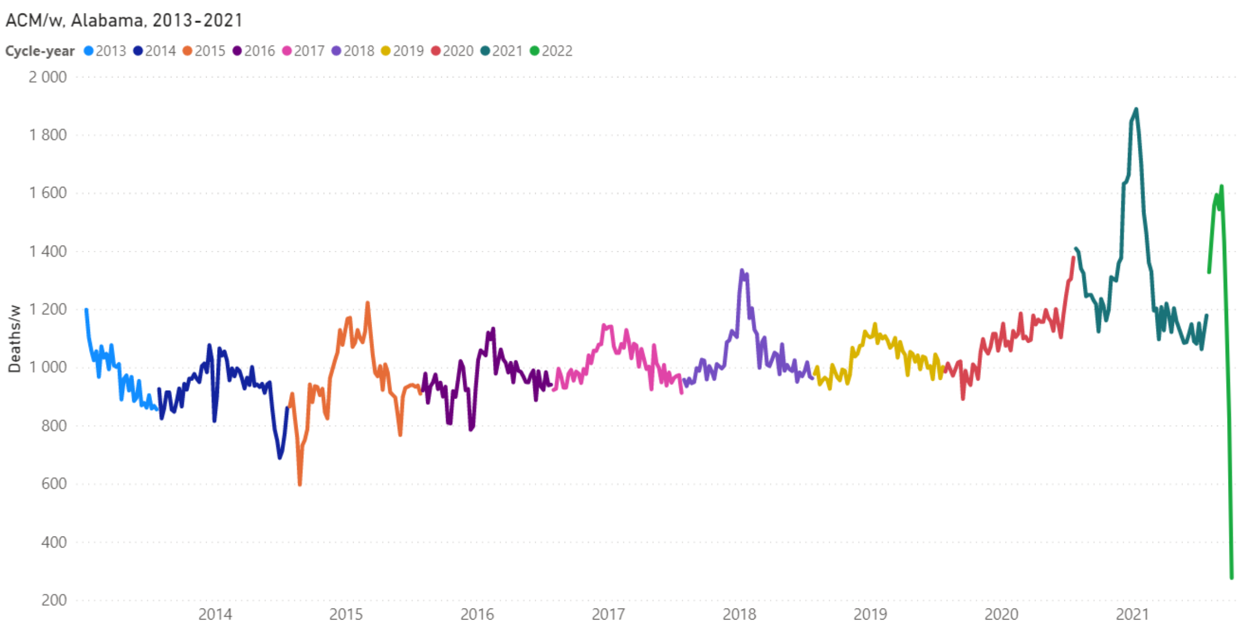
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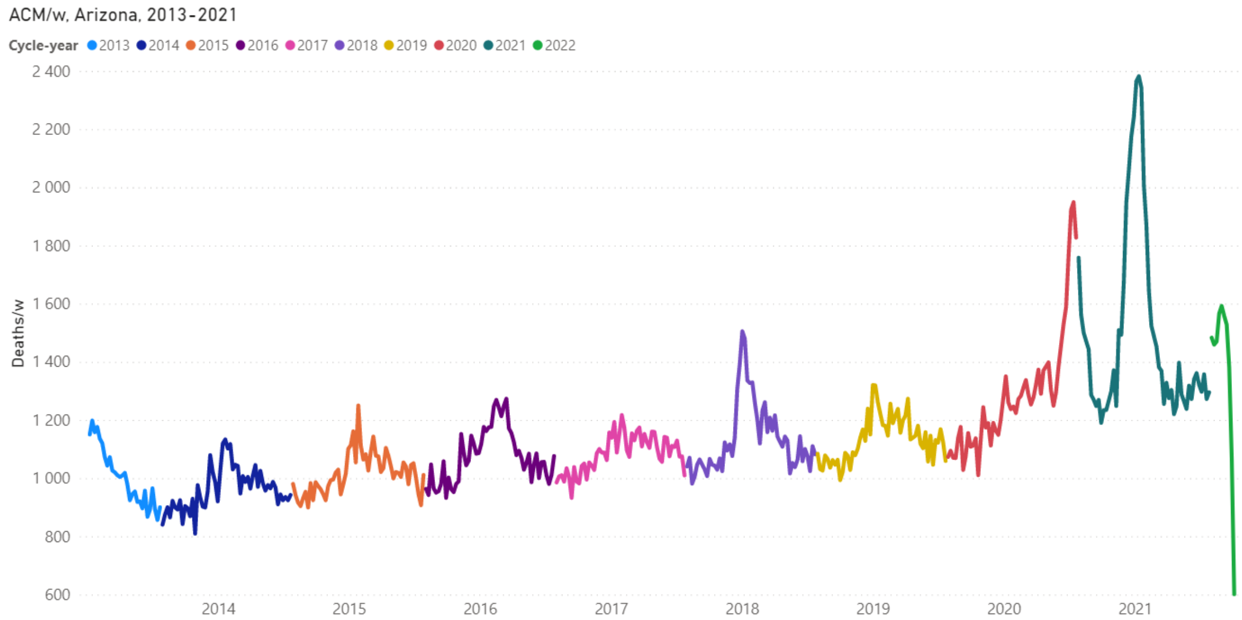
Appendix:

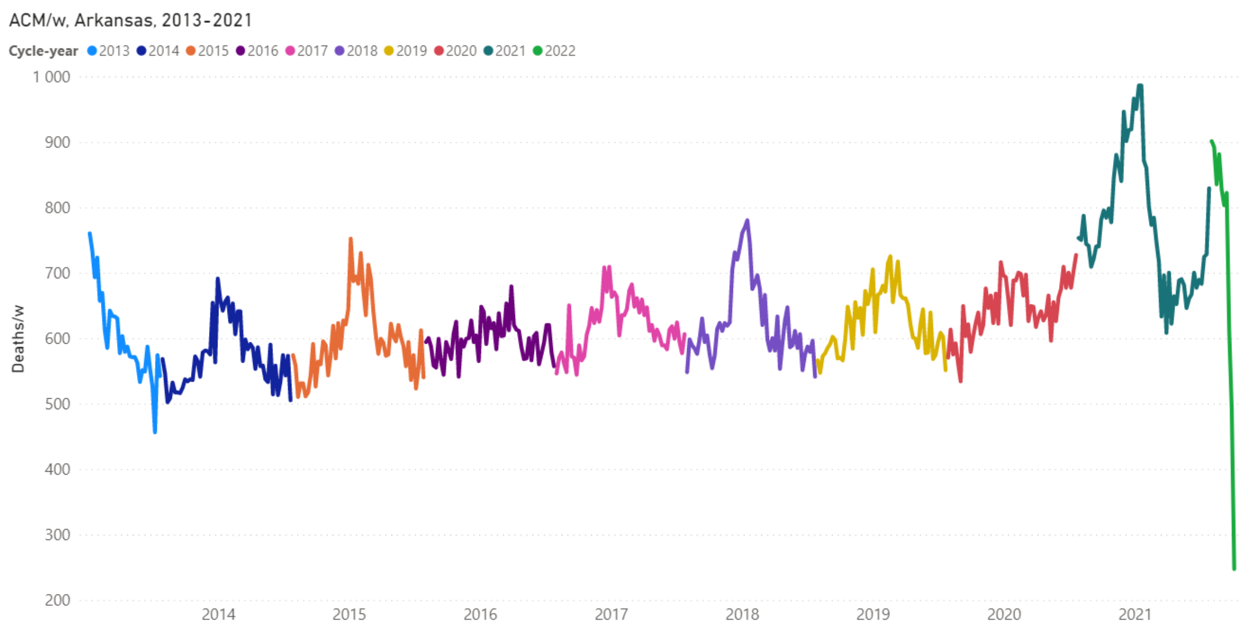
ACM/w, 2013-2021, with colour-differentiated cycle-years, for all the individual states of continental USA

The following graphs represent the all-cause mortality by week in each state of the continental USA from 2013 to 2021. Data are displayed from week-1 of 2013 to week-40 of 2021 (last available data point at the date of access, unless otherwise stated). The different colours are for the different cycle-years. The cycle-year starts on week-31 of a calendar-year (beginning of August) and ends on week-30 of the next calendar-year (end of July). Cycle-years 2013 and 2022 are then not completed. Data were retrieved from CDC (CDC, 2021a), as described in Table 1 of section 2 of the article.

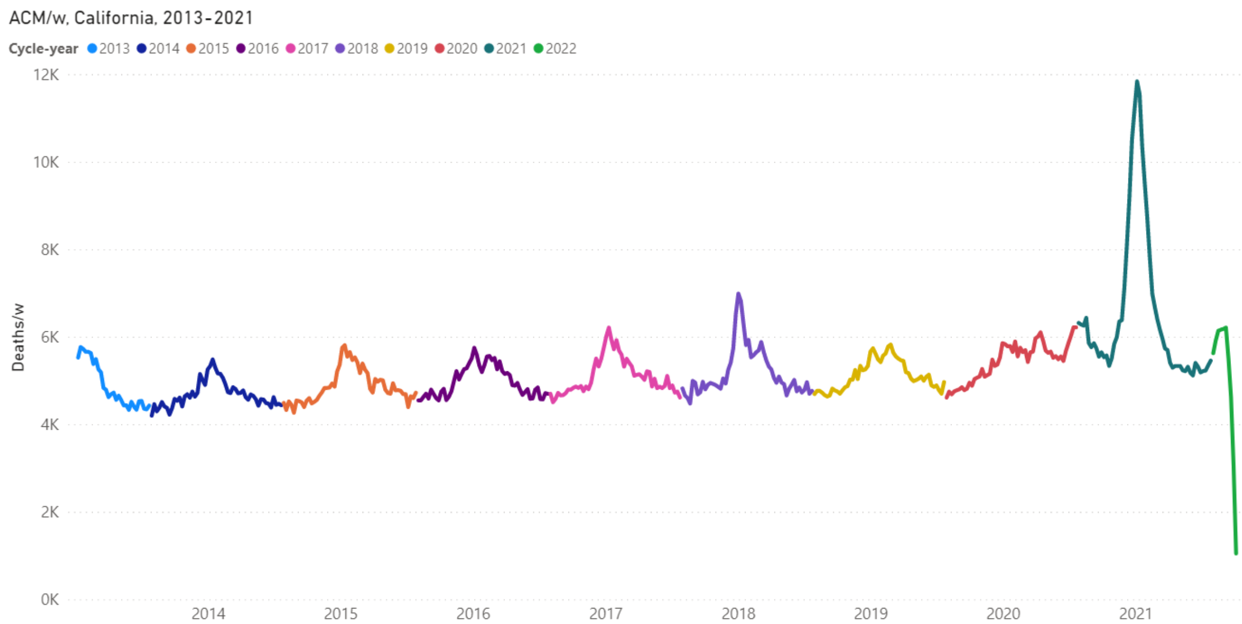
The 49 continental USA states, including District of Columbia and excluding Alaska and Hawaii, are presented by alphabetical order.

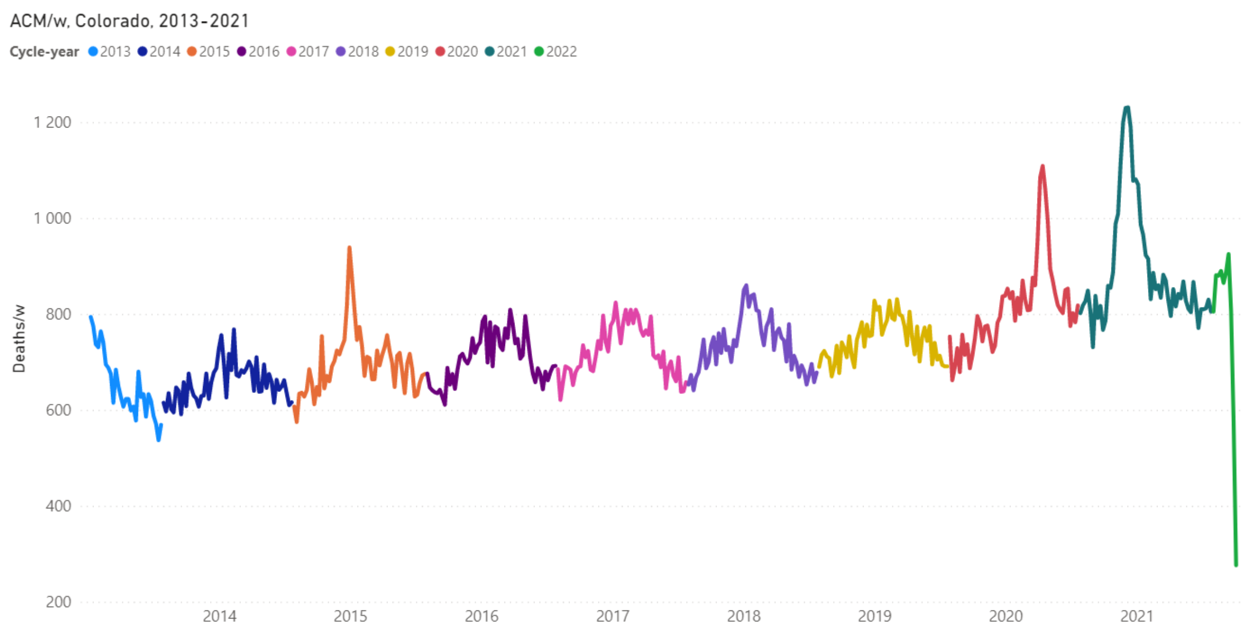


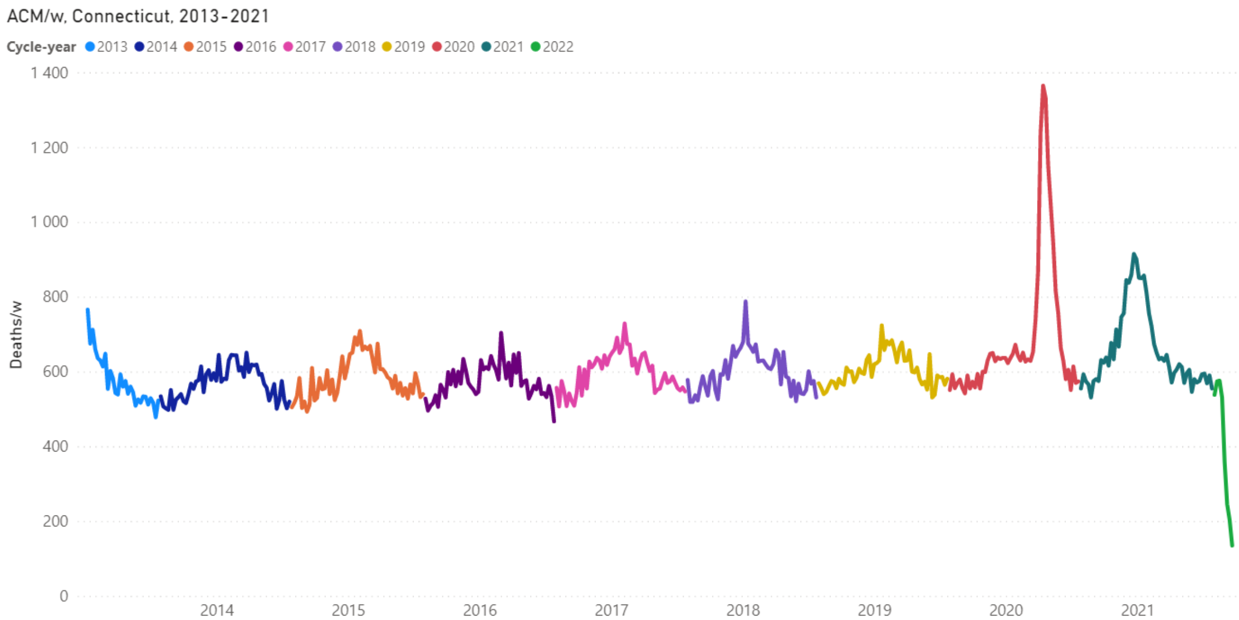

ACM/w, Arizona, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
2 400
2 200
2 000
1 800
1 600
1 400
1 200
1 000
800
600
Deaths/w
2014 2015 2016 2017 2018 2019 2020 2021


ACM/w, Arkansas, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
1 000
900
800
700
600
500
400
300
200
Deaths/w
2014 2015 2016 2017 2018 2019 2020 2021

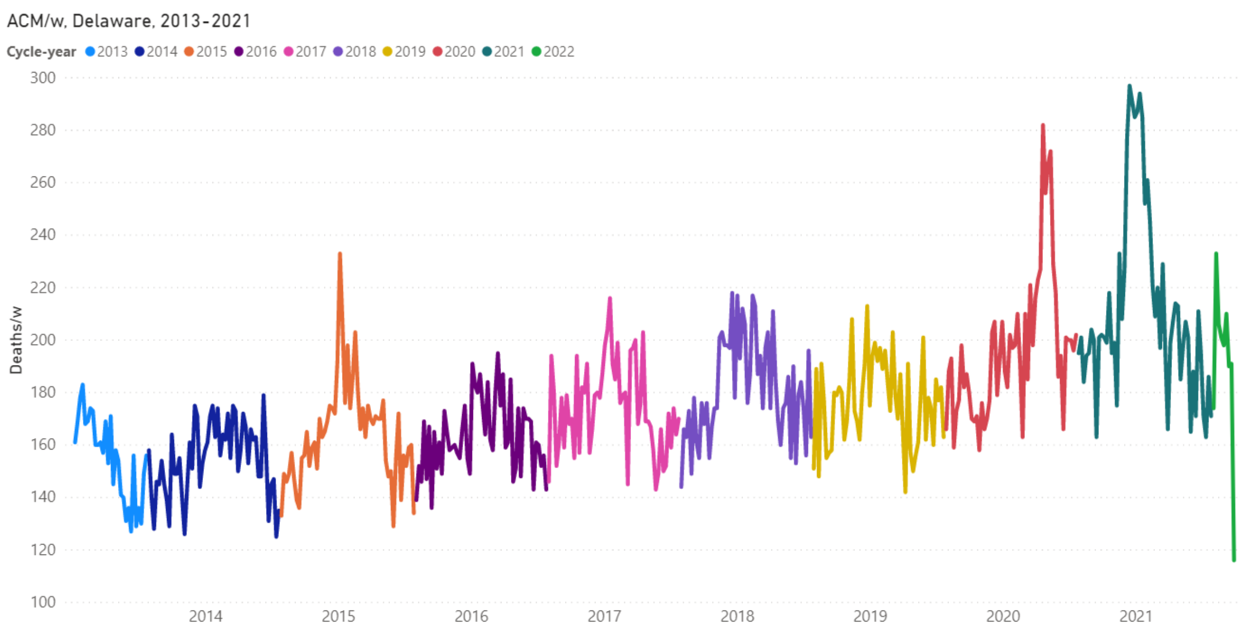
563


ACM/w, California, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
12K
10K
8K
6K
4K
2K
0K
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021


ACM/w, Colorado, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
1 200
1 000
800
600
400
200
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021

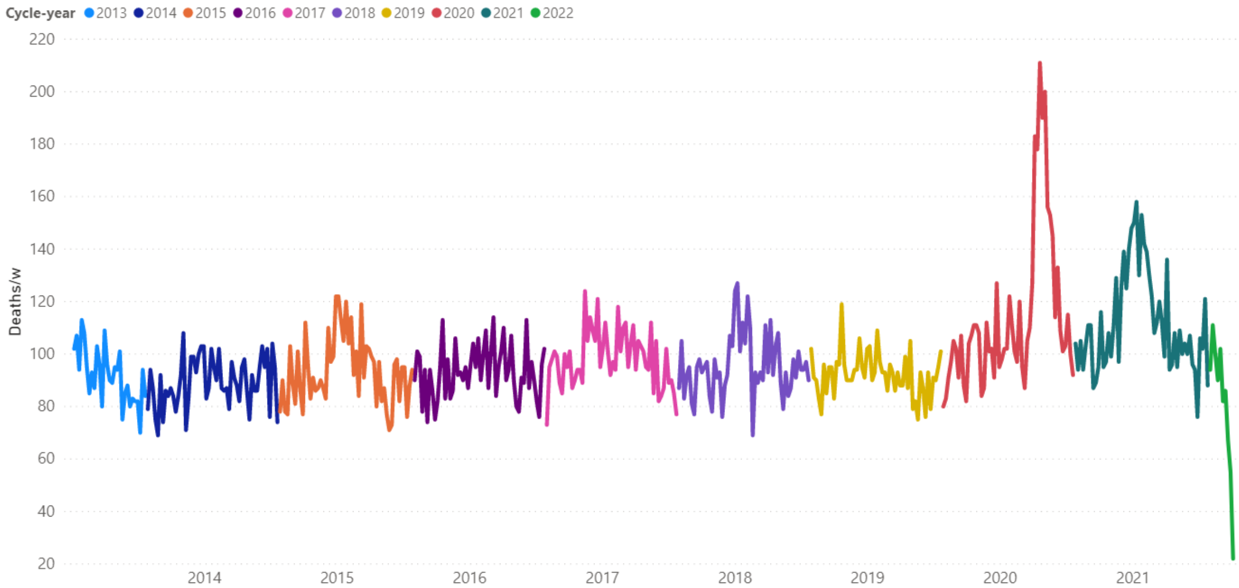


The last data point of Connecticut is week-38 of 2021.

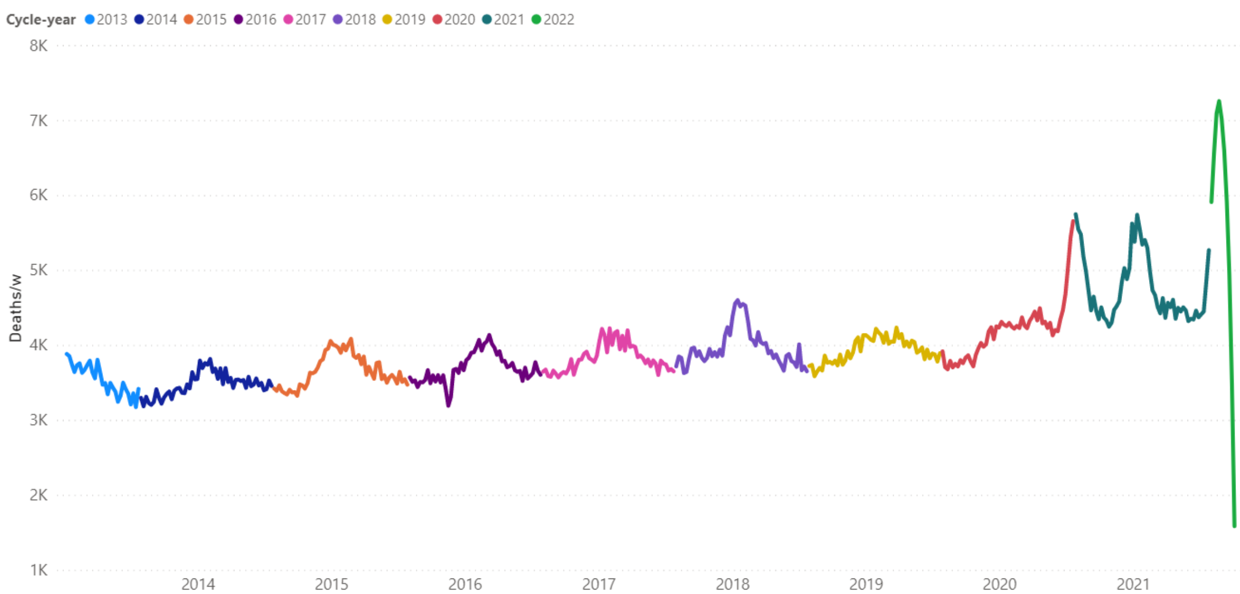


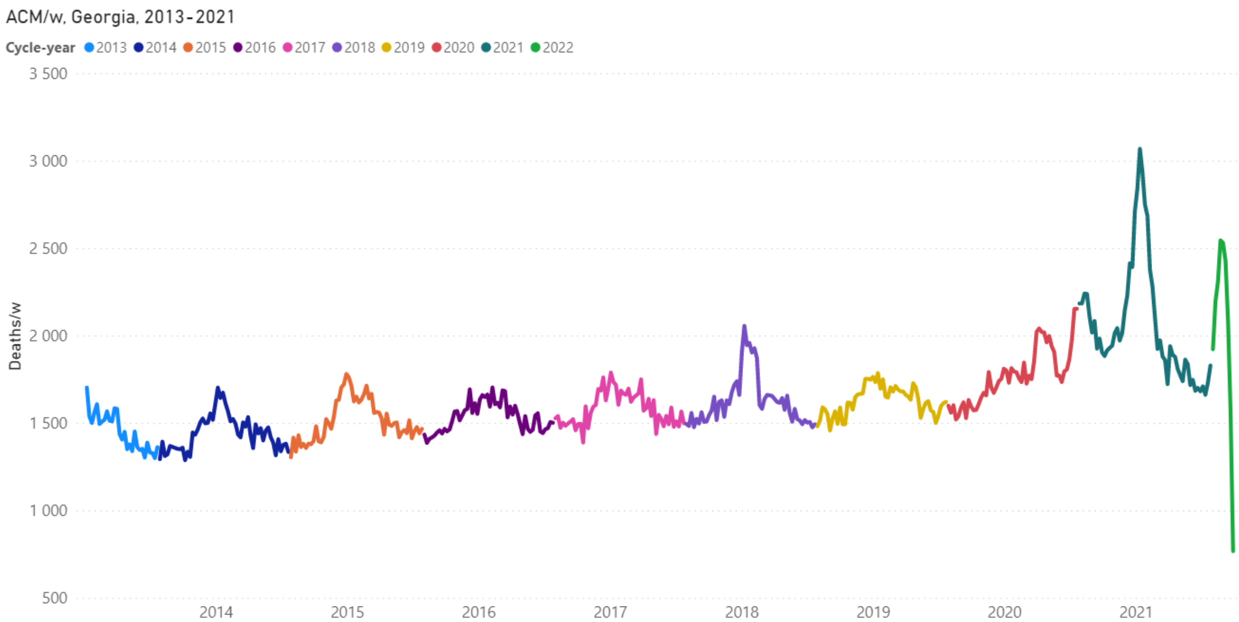
The last data point of Delaware is week-39 of 2021.

ACM/w, District of Columbia, 2013-2021

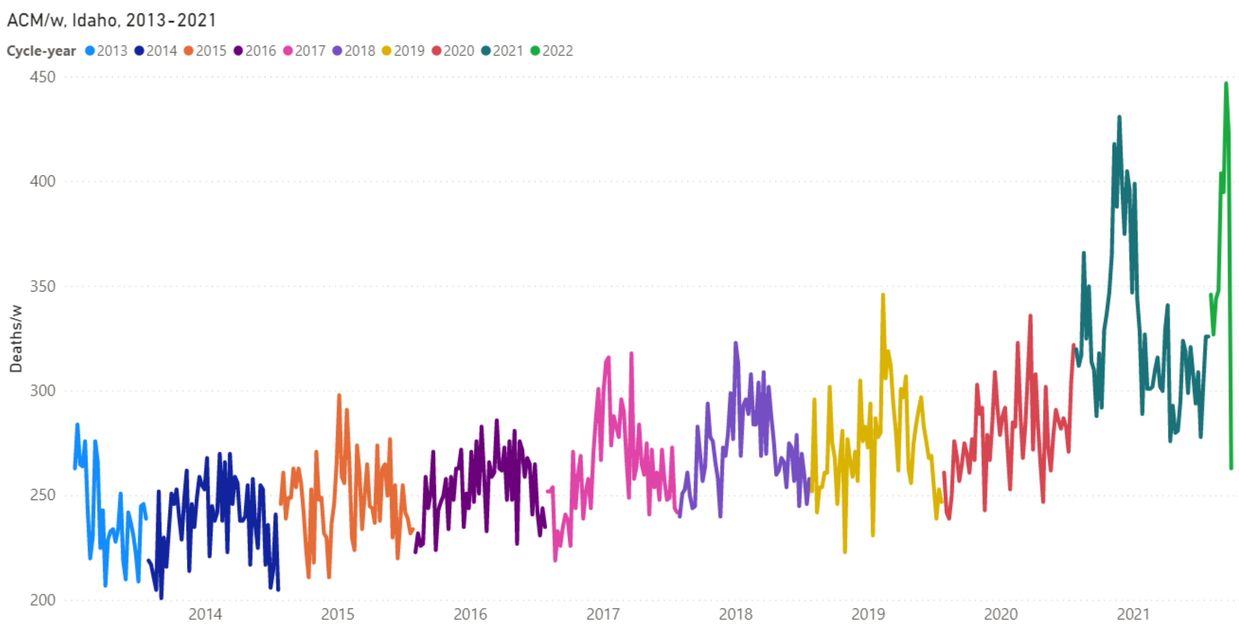


ACM/w, Florida, 2013-2021

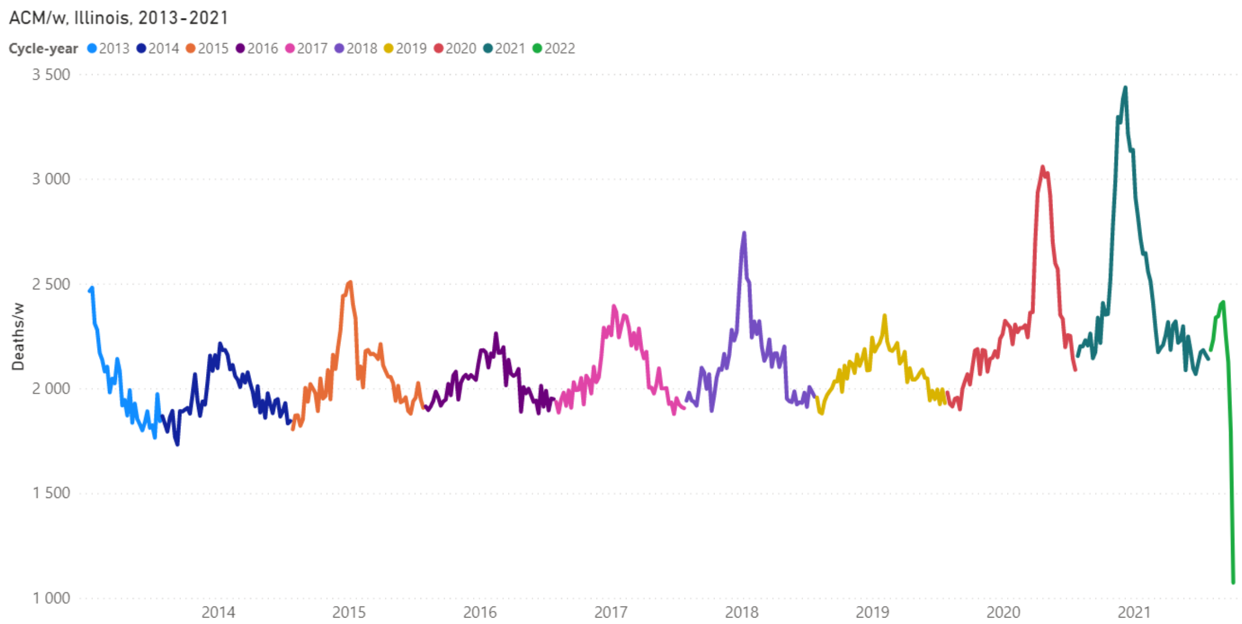


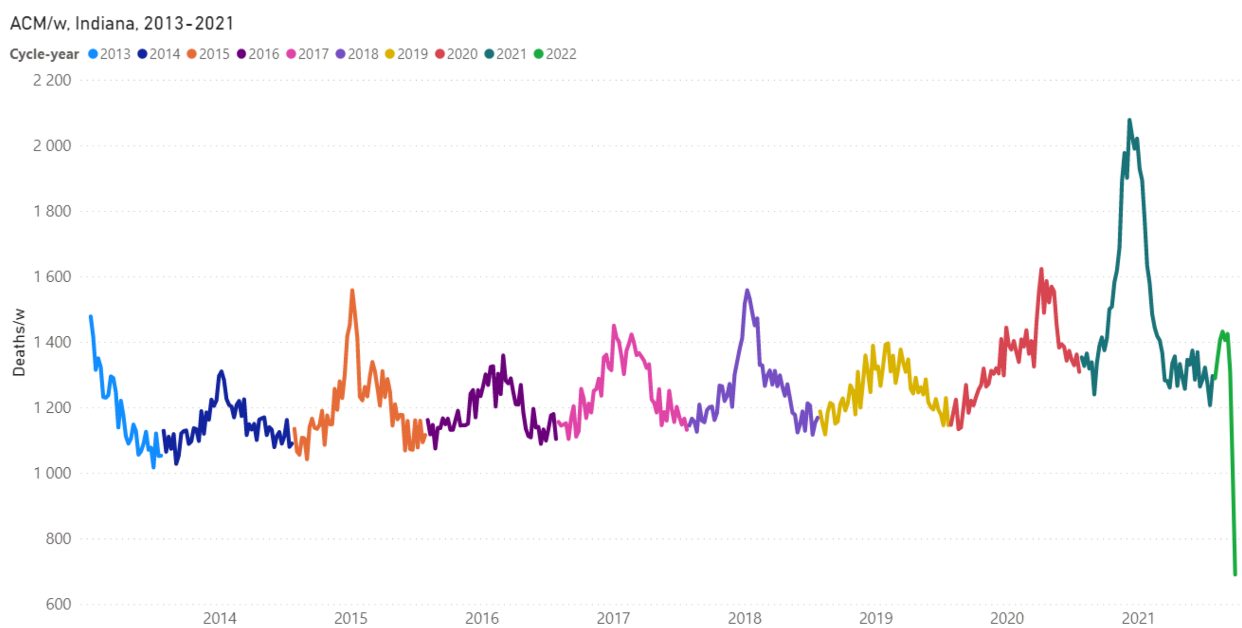


The last data point of Georgia is week-39 of 2021.

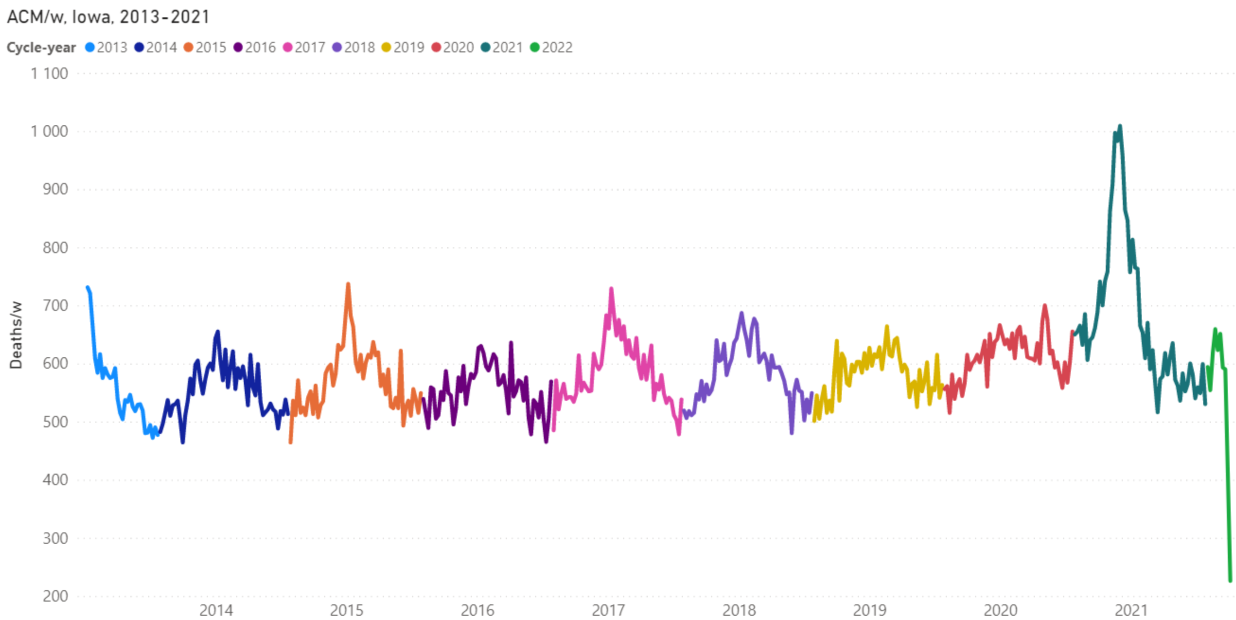


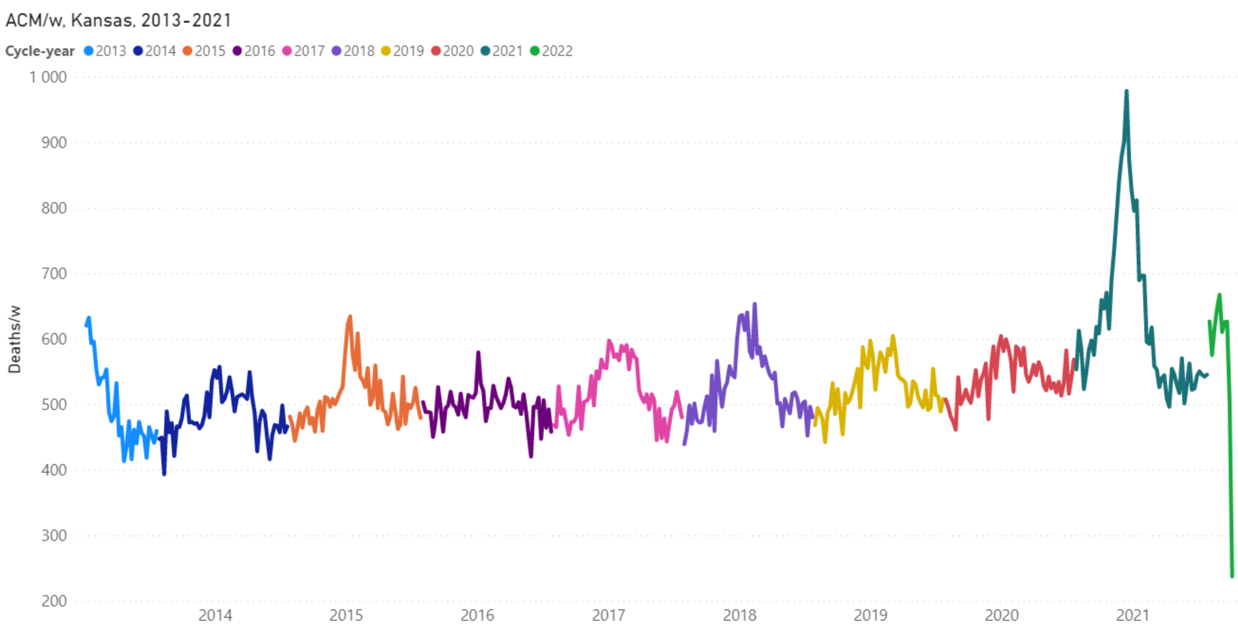
The last data point of Idaho is week-39 of 2021.

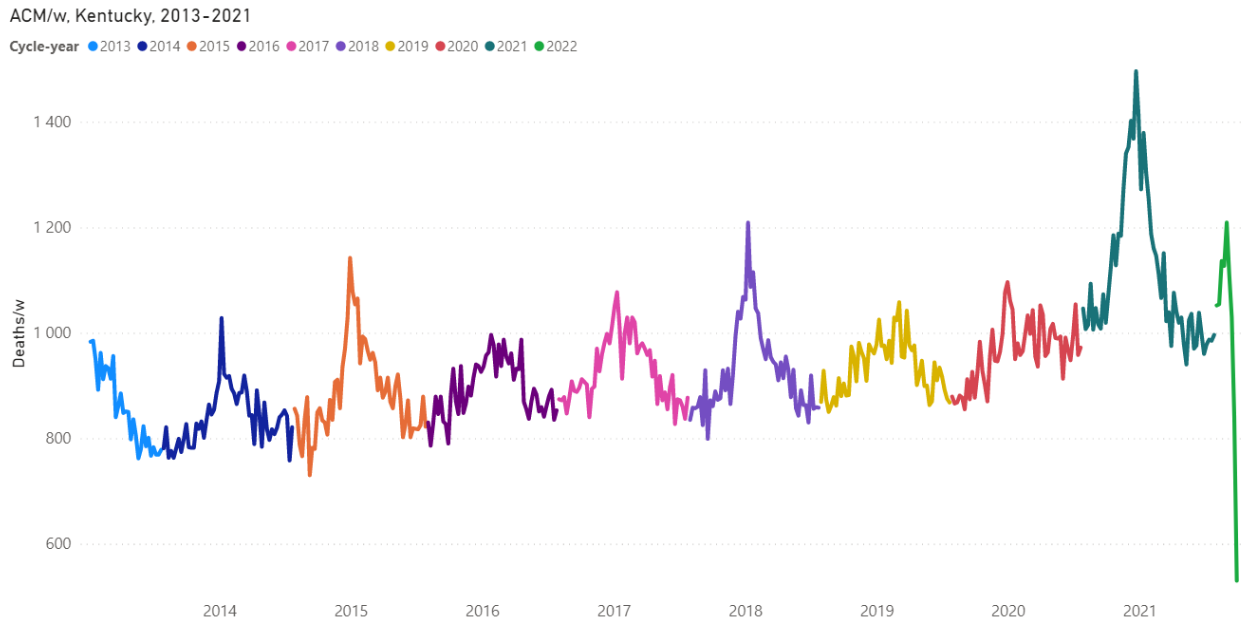




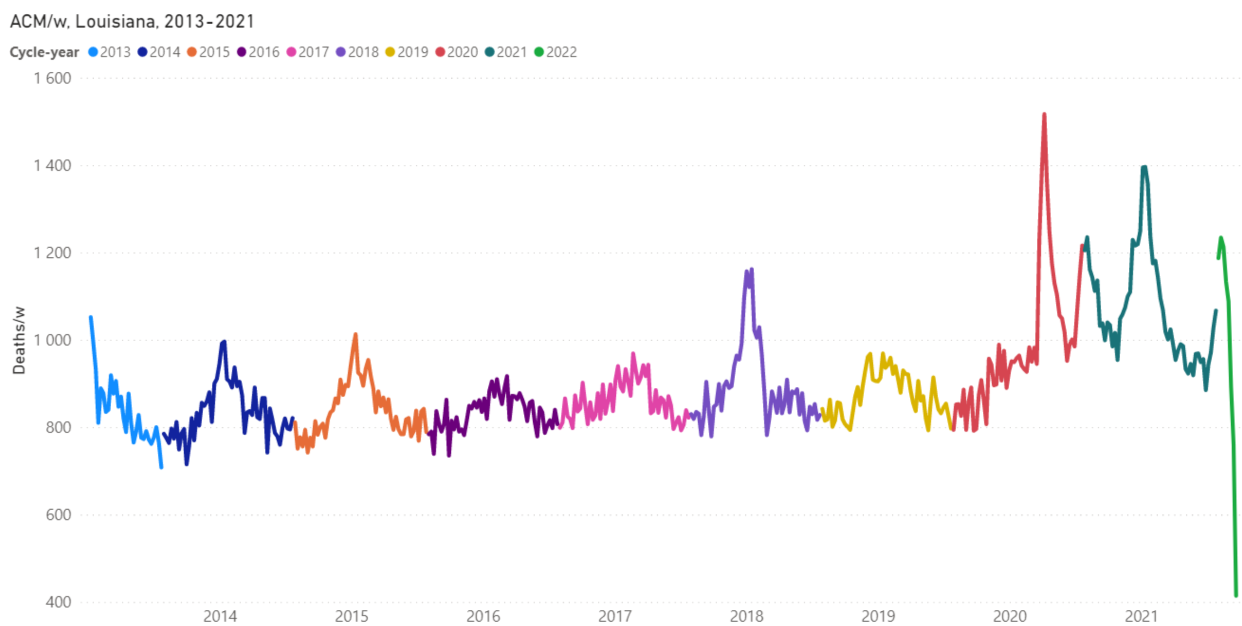
The last data point of Indiana is week-39 of 2021.


ACM/w, Iowa, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
1 100
1 000
900
800
700
600
500
400
300
200
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021

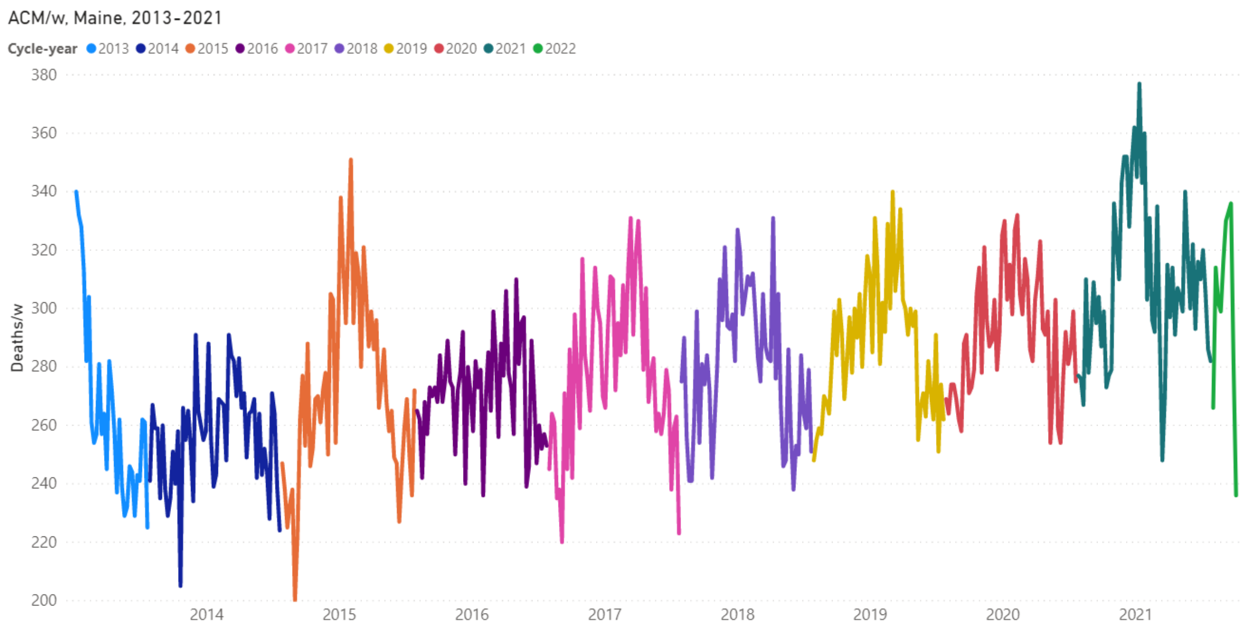

ACM/w, Kansas, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
1 000
900
800
700
600
500
400
300
200
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021

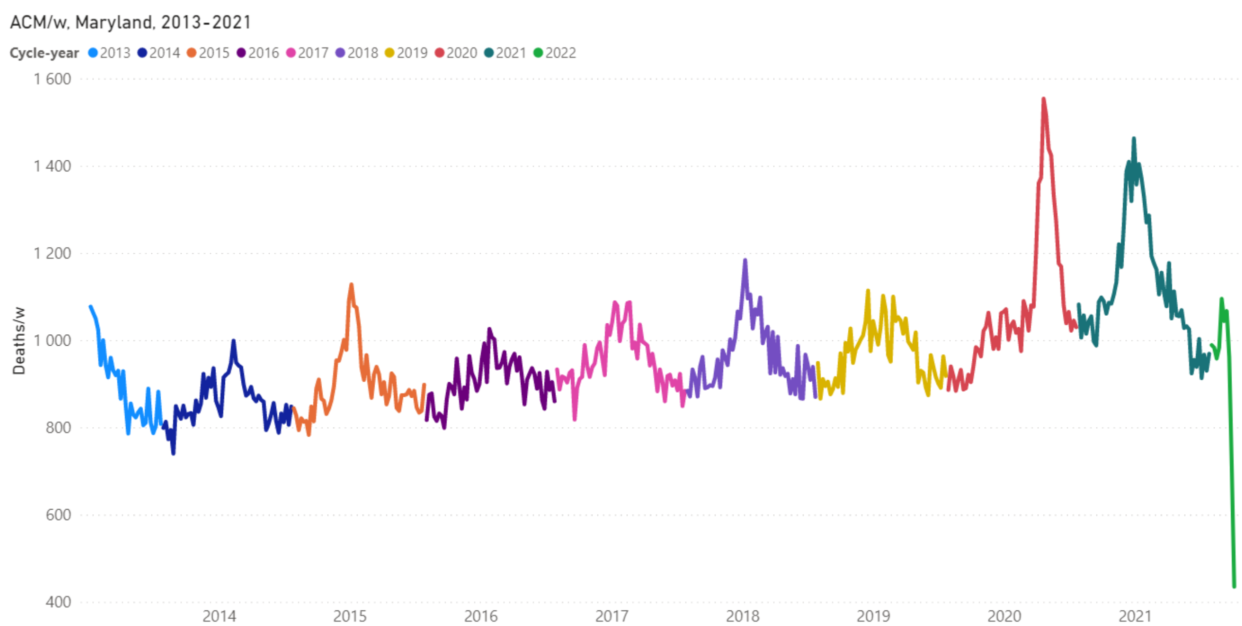


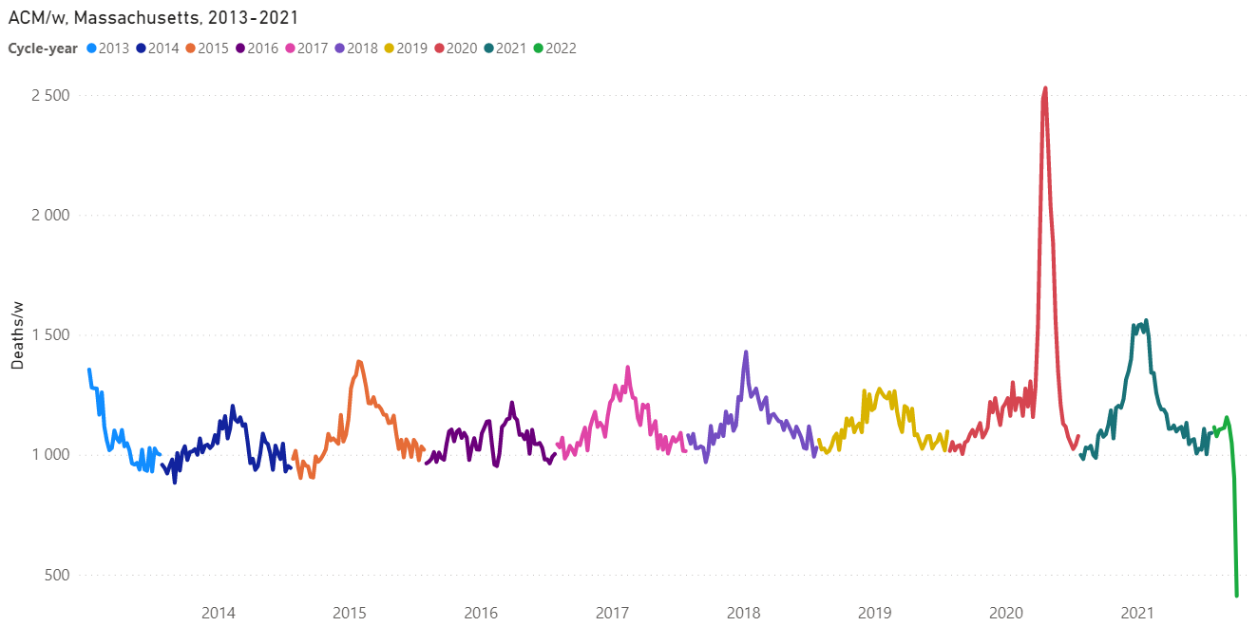
The last data point of Kentucky is week-39 of 2021.

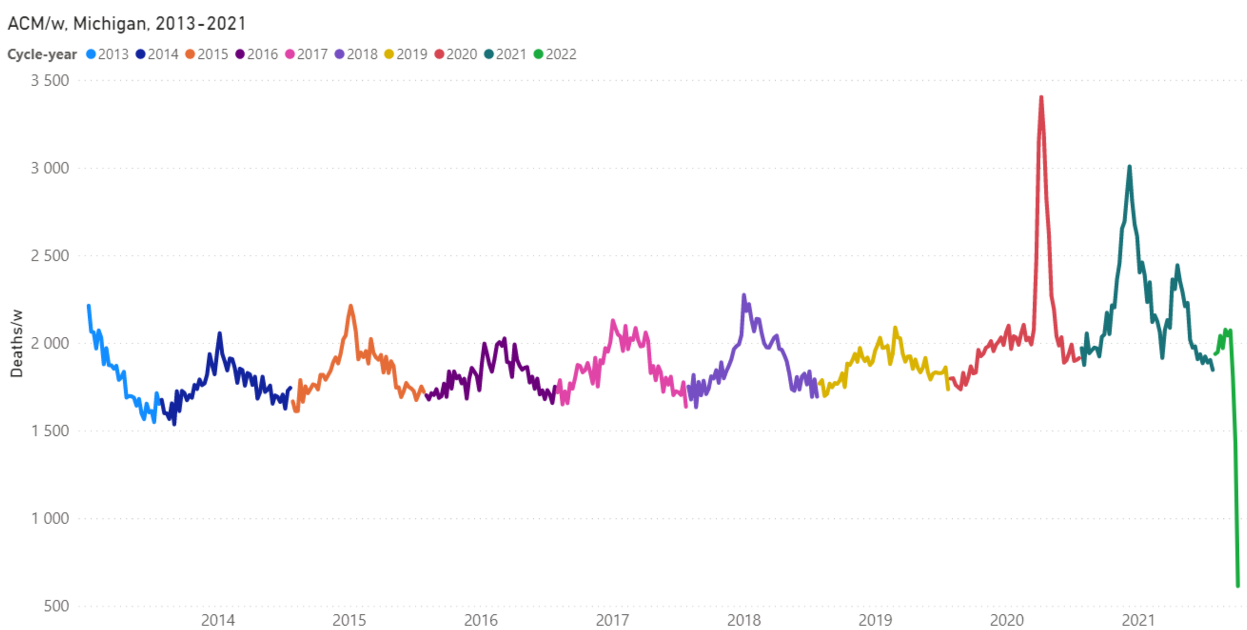


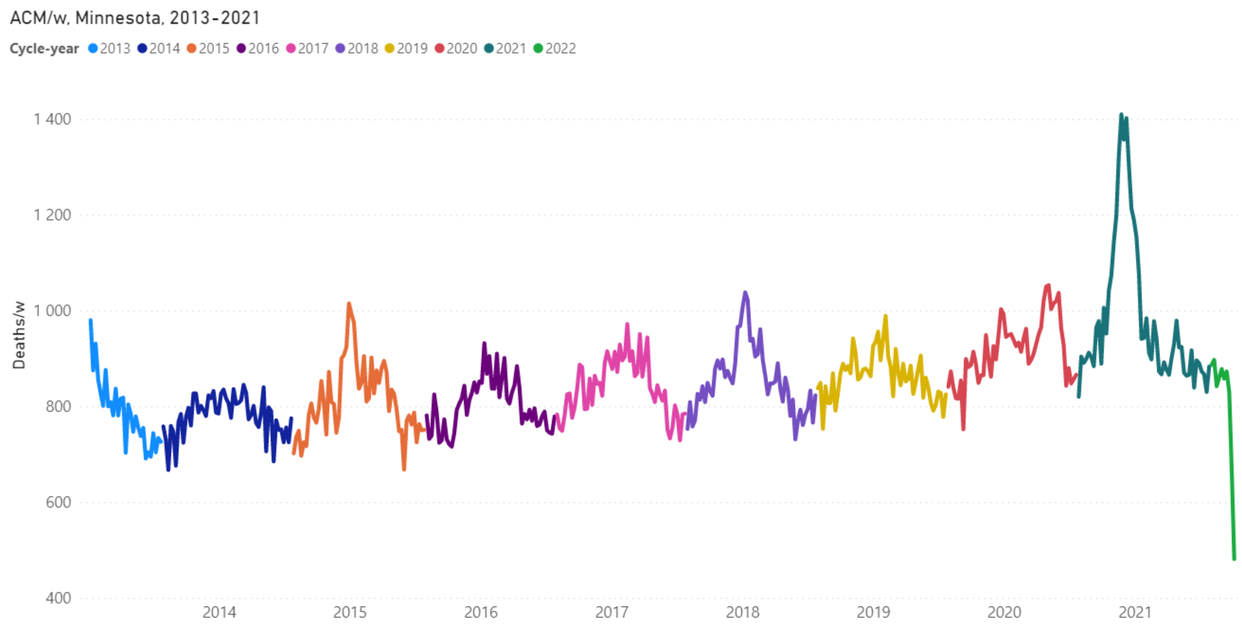
The last data point of Louisiana is week-38 of 2021.

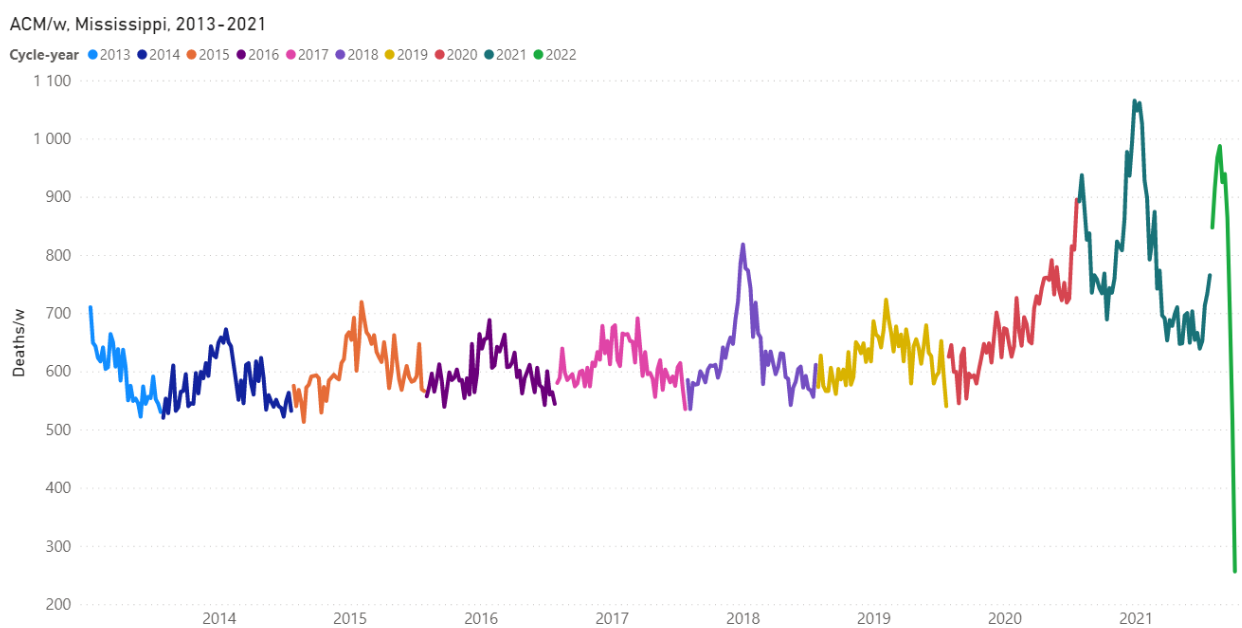

ACM/w, Maine, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
380
360
340
320
300
280
260
240
220
200
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021

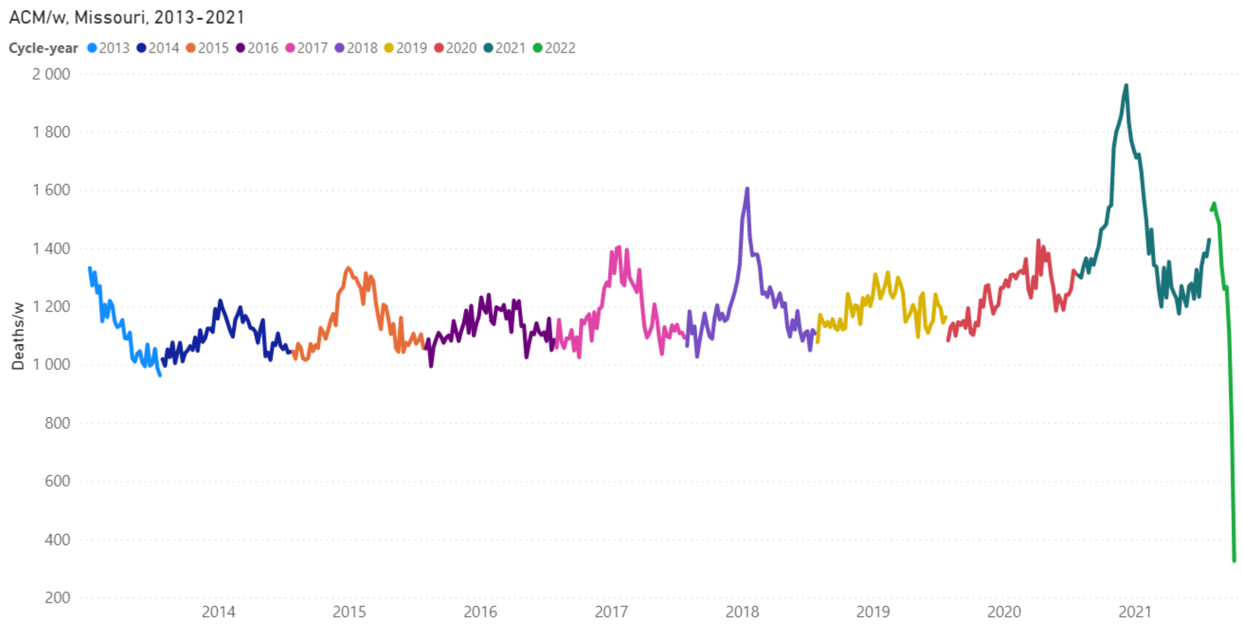

ACM/w, Maryland, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
1 600
1 400
1 200
1 000
800
600
400
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021

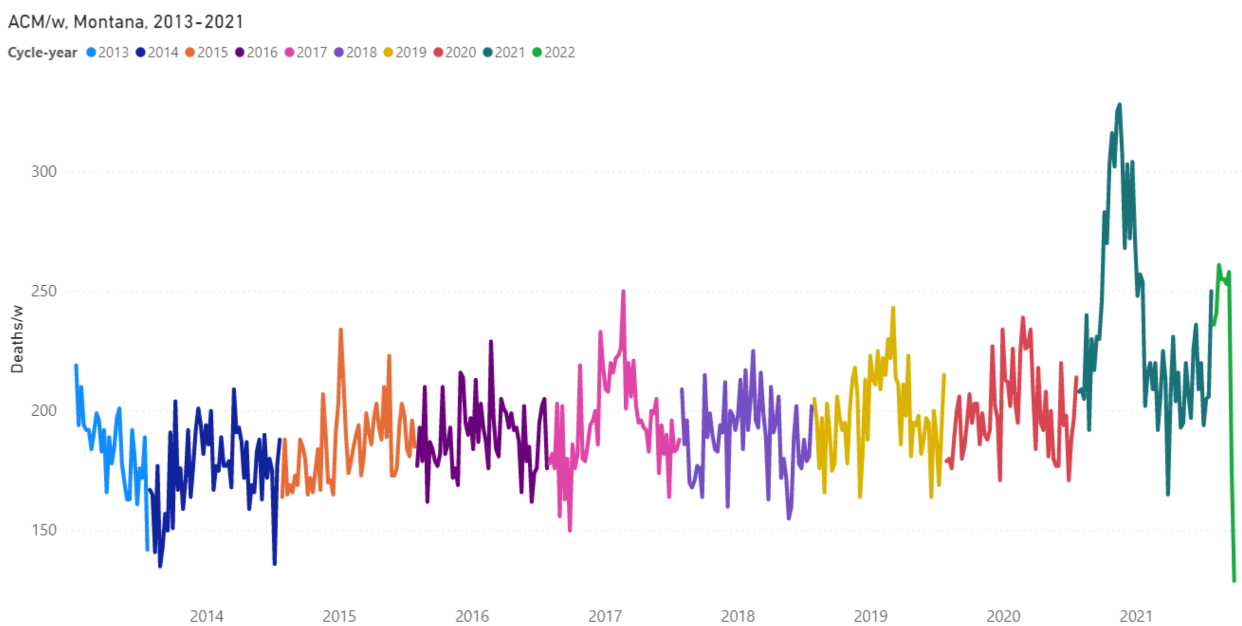

ACM/w, Massachusetts, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
Deaths/w
2 500
2 000
1 500
1 000
500
2014
2015
2016
2017
2018
2019
2020
2021


ACM/w, Michigan, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
Deaths/w
3 500
3 000
2 500
2 000
1 500
1 000
500
2014
2015
2016
2017
2018
2019
2020
2021

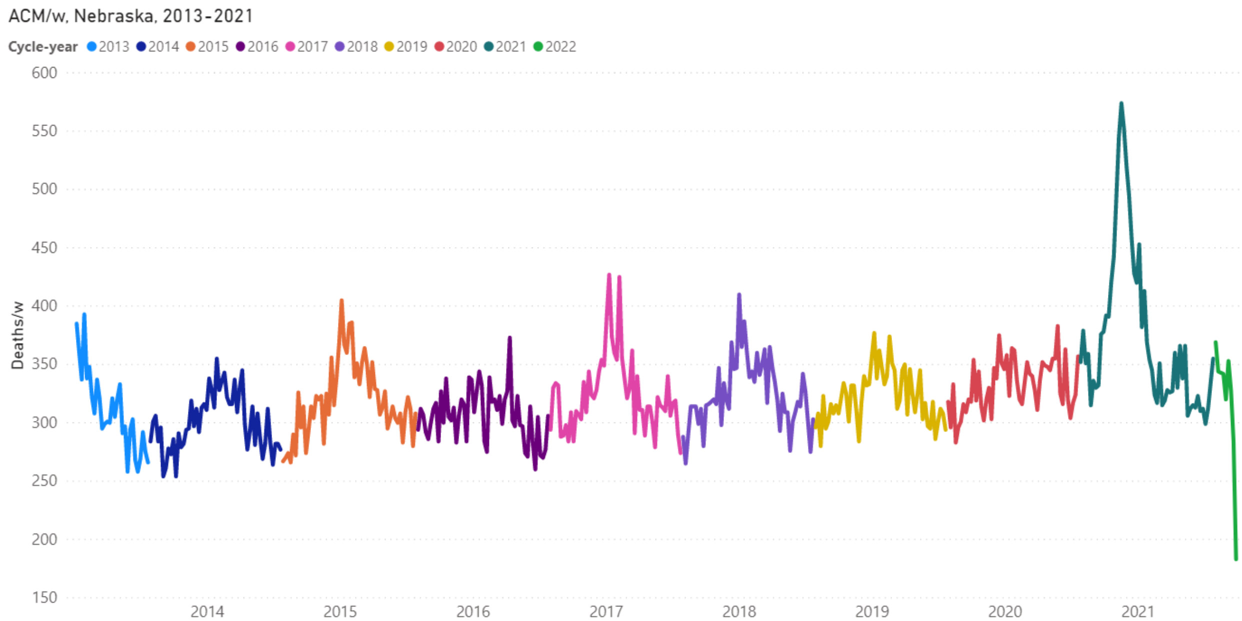

ACM/w, Minnesota, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
Deaths/w
1 400
1 200
1 000
800
600
400
2014
2015
2016
2017
2018
2019
2020
2021


ACM/w, Mississippi, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
Deaths/w
1 100
1 000
900
800
700
600
500
400
300
200
2014
2015
2016
2017
2018
2019
2020
2021

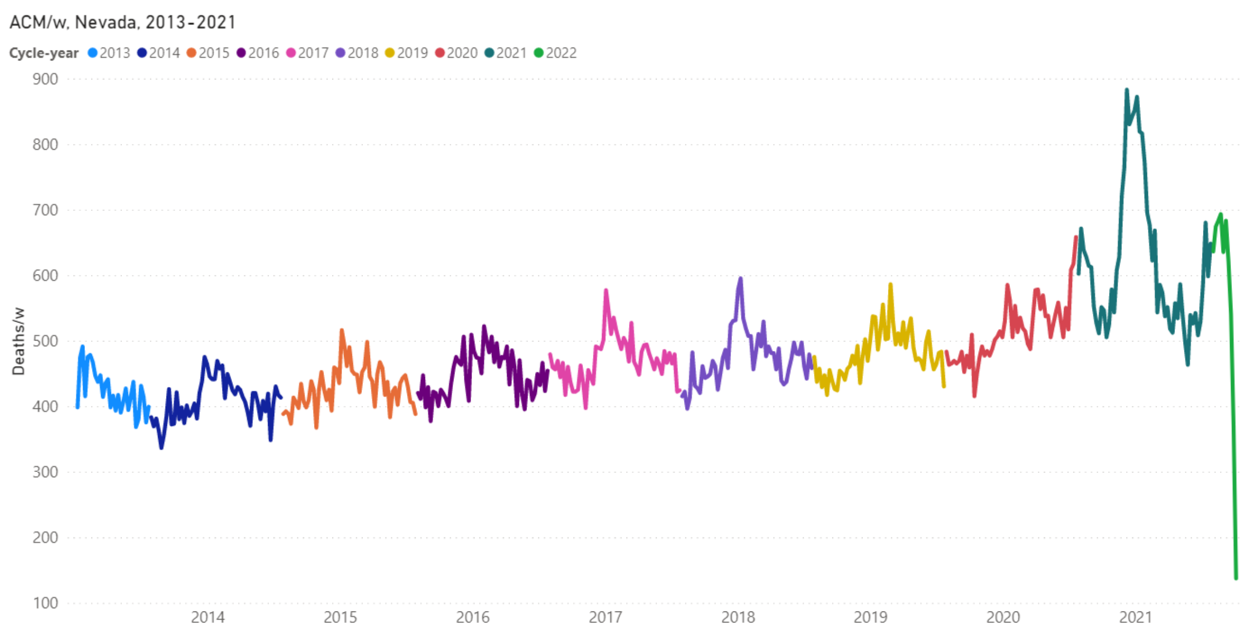


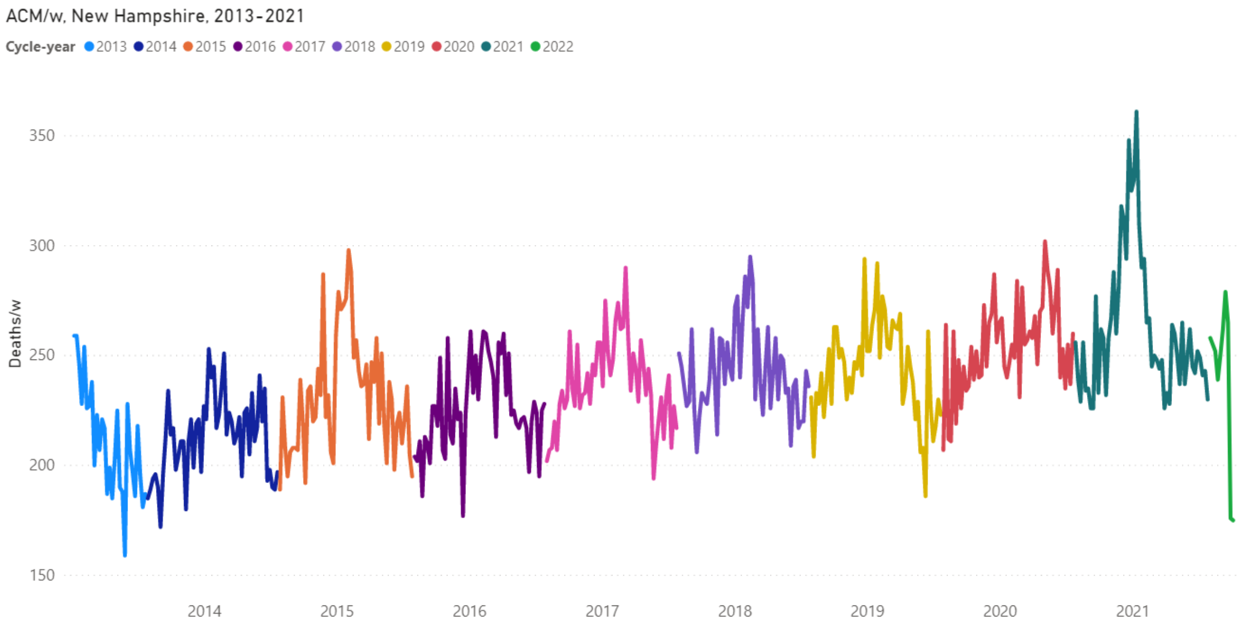


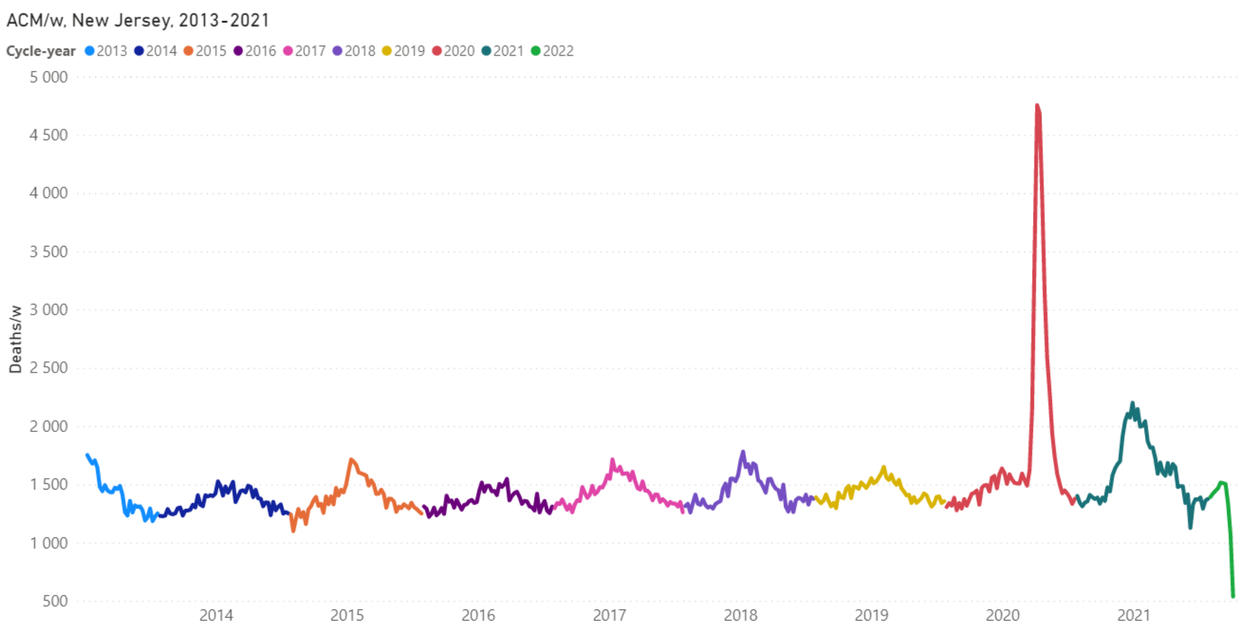
The last data point of Montana is week-39 of 2021.



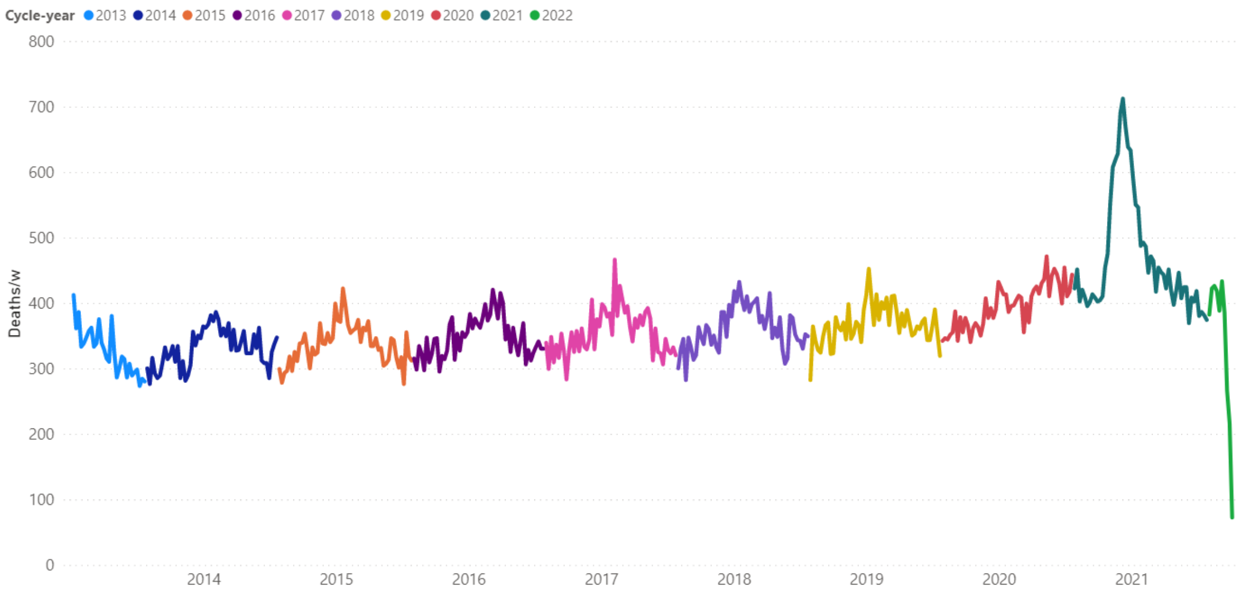
The last data point of Nebraska is week-39 of 2021.



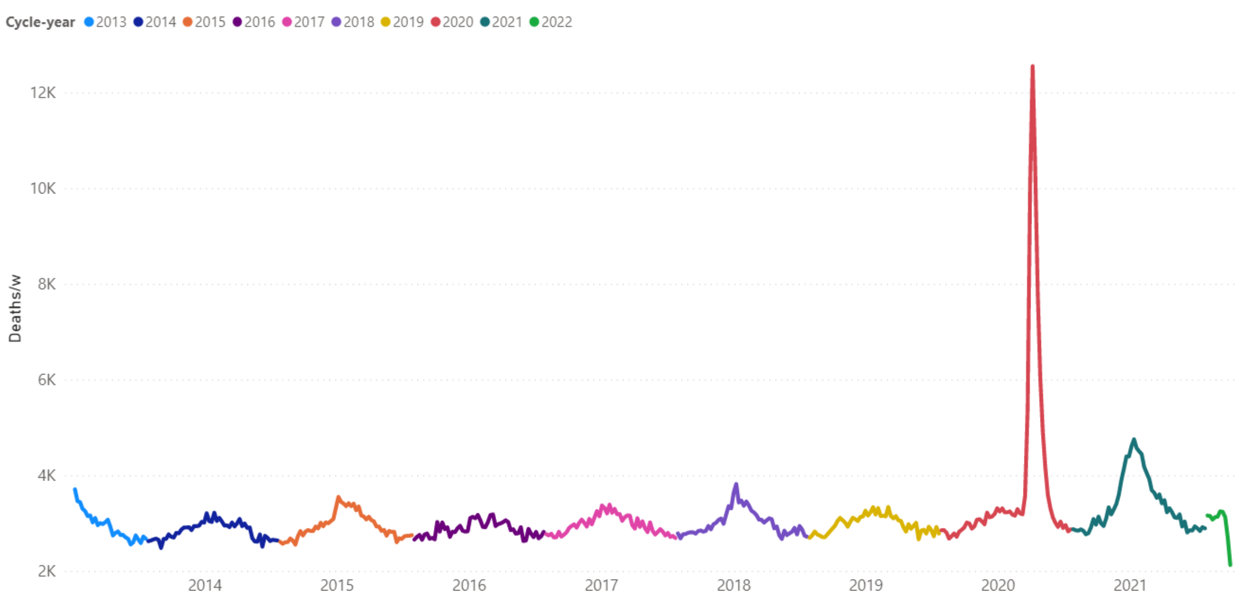

ACM/w, New Hampshire, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
Deaths/w
350
300
250
200
150
2014
2015
2016
2017
2018
2019
2020
2021

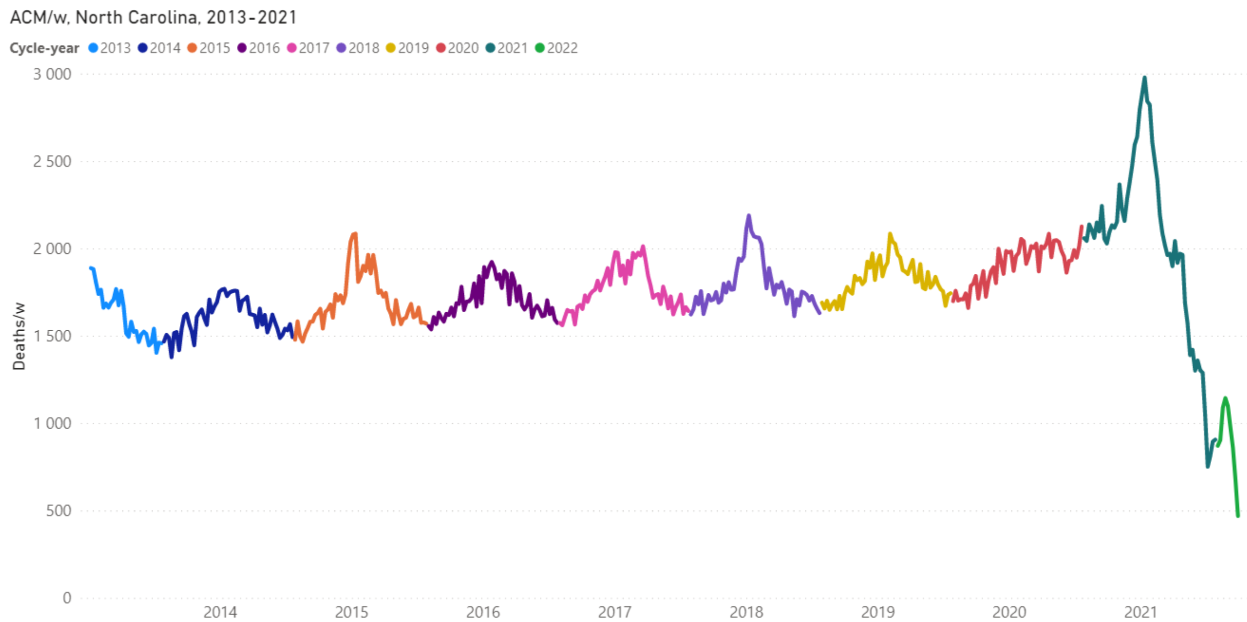

ACM/w, New Jersey, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
Deaths/w
5 000
4 500
4 000
3 500
3 000
2 500
2 000
1 500
1 000
500
2014
2015
2016
2017
2018
2019
2020
2021

ACM/w, New Mexico, 2013-2021

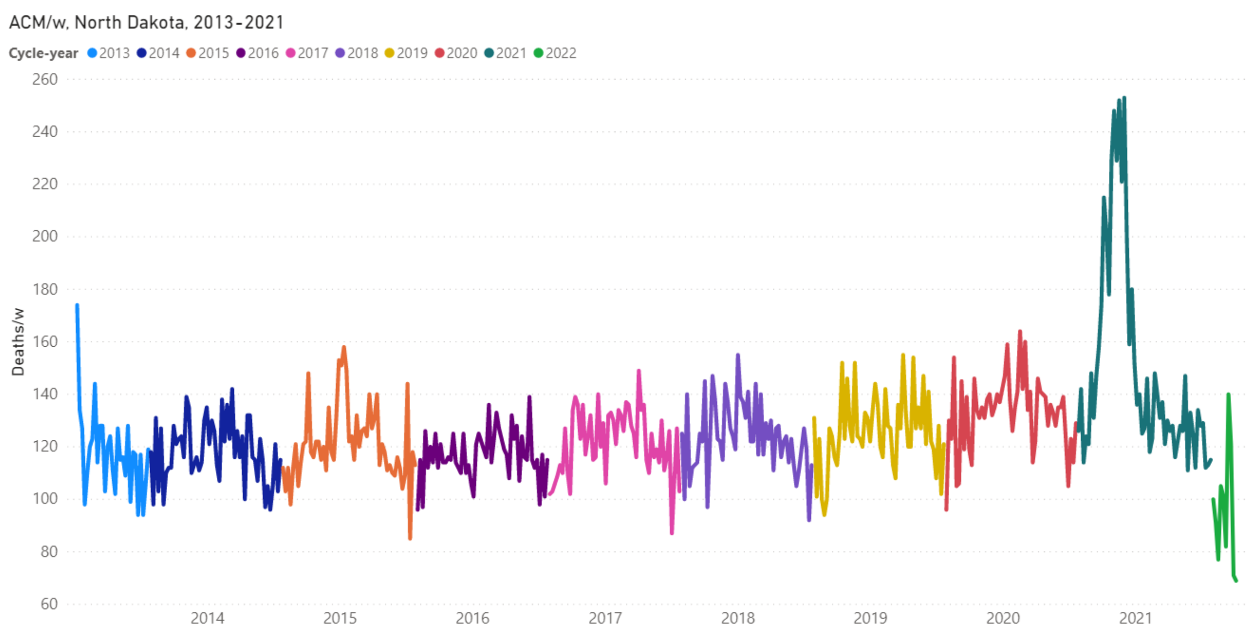


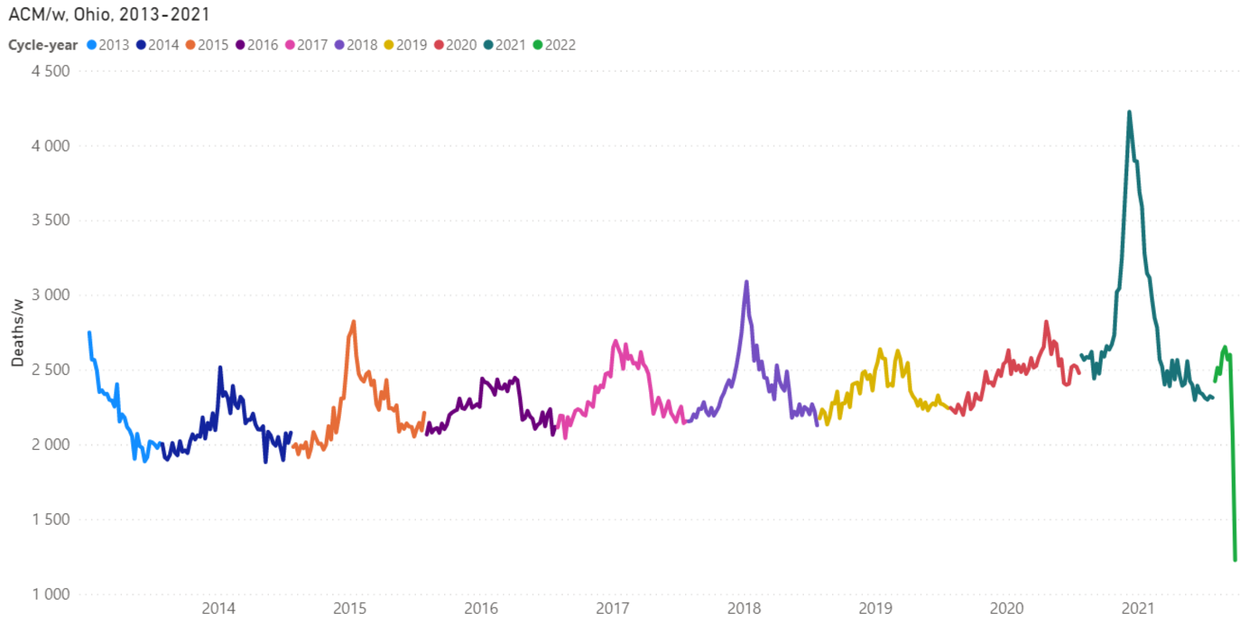
ACM/w, New York, 2013-2021



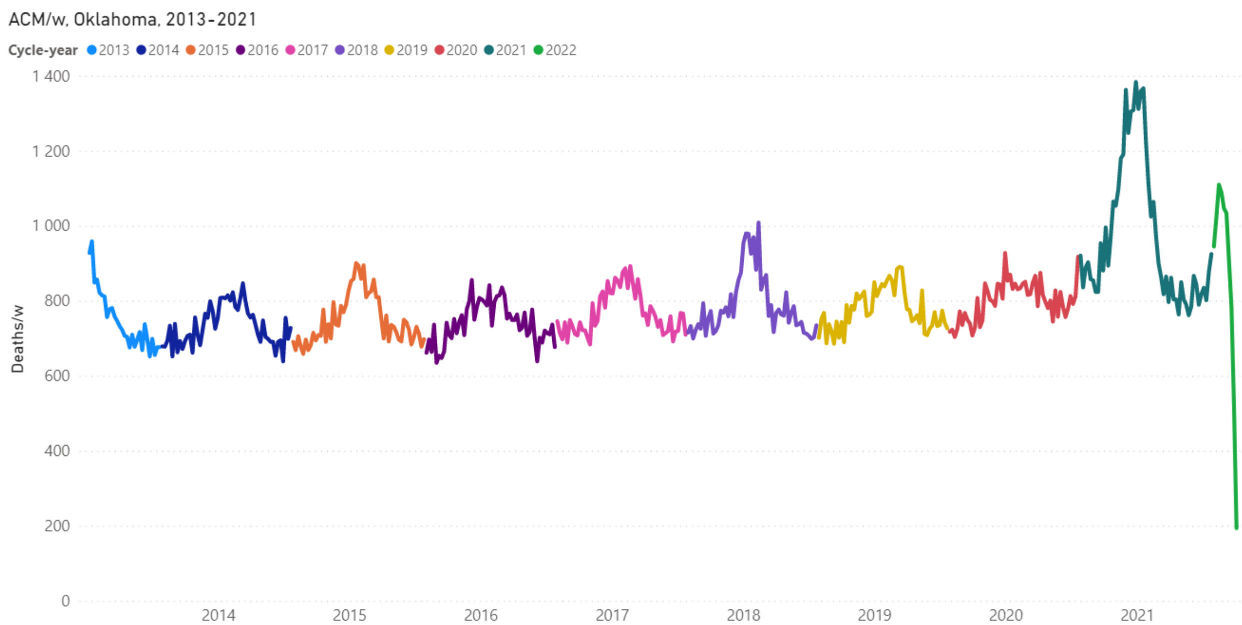


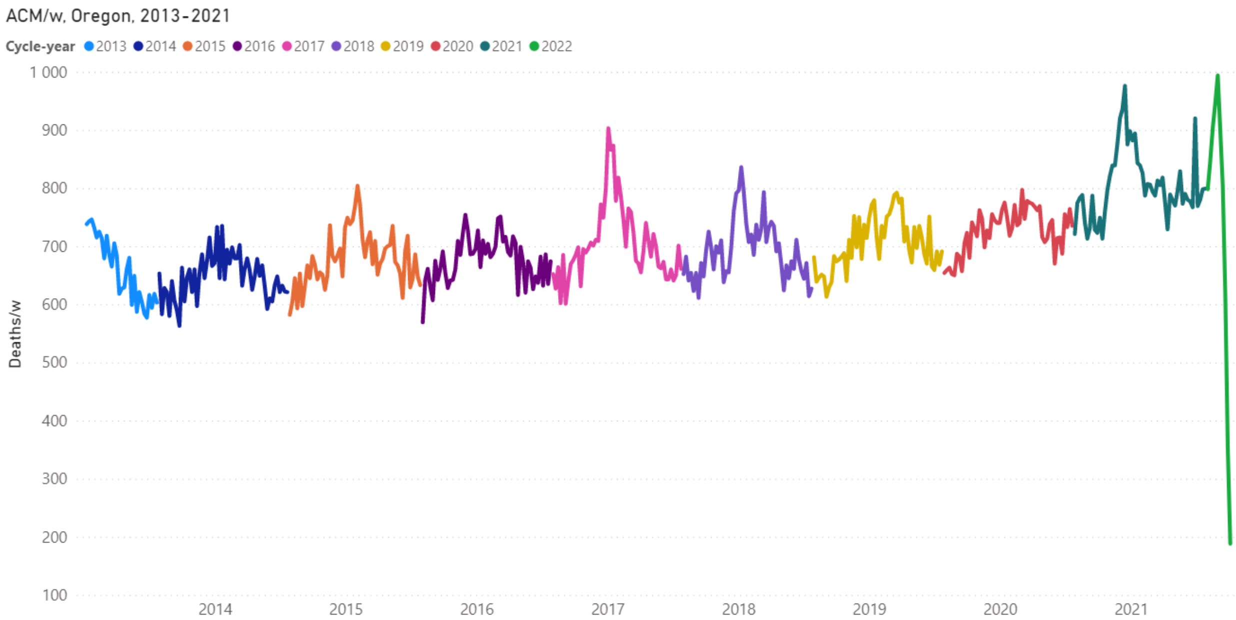
The last data point of North Carolina is week-39 of 2021.

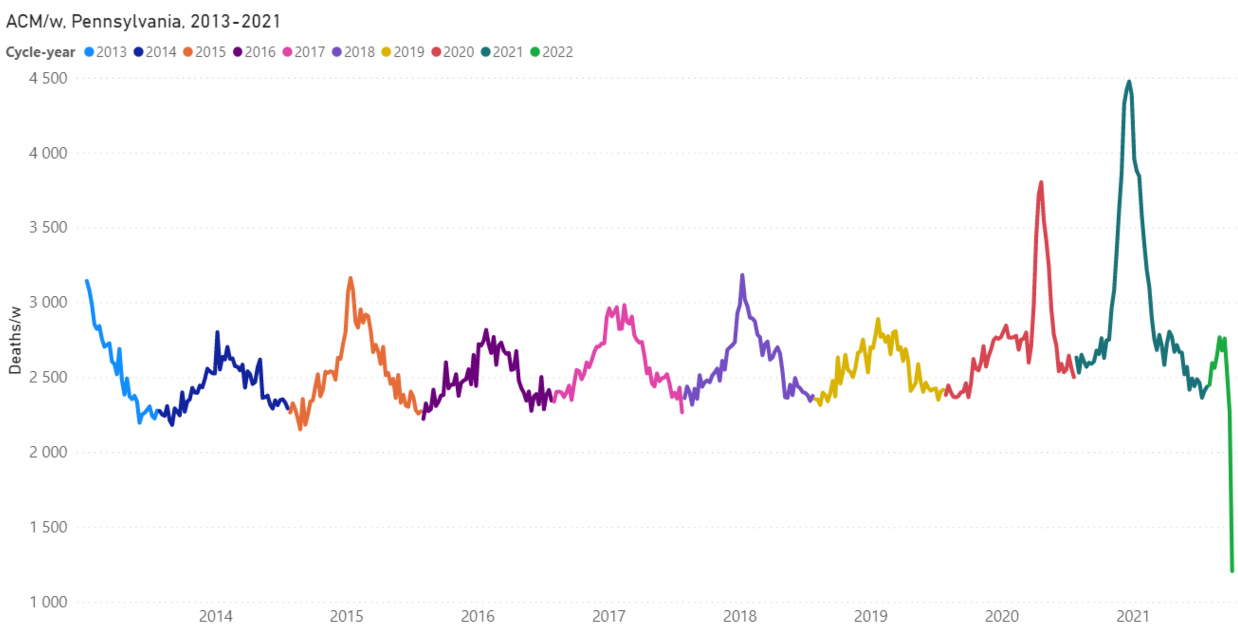


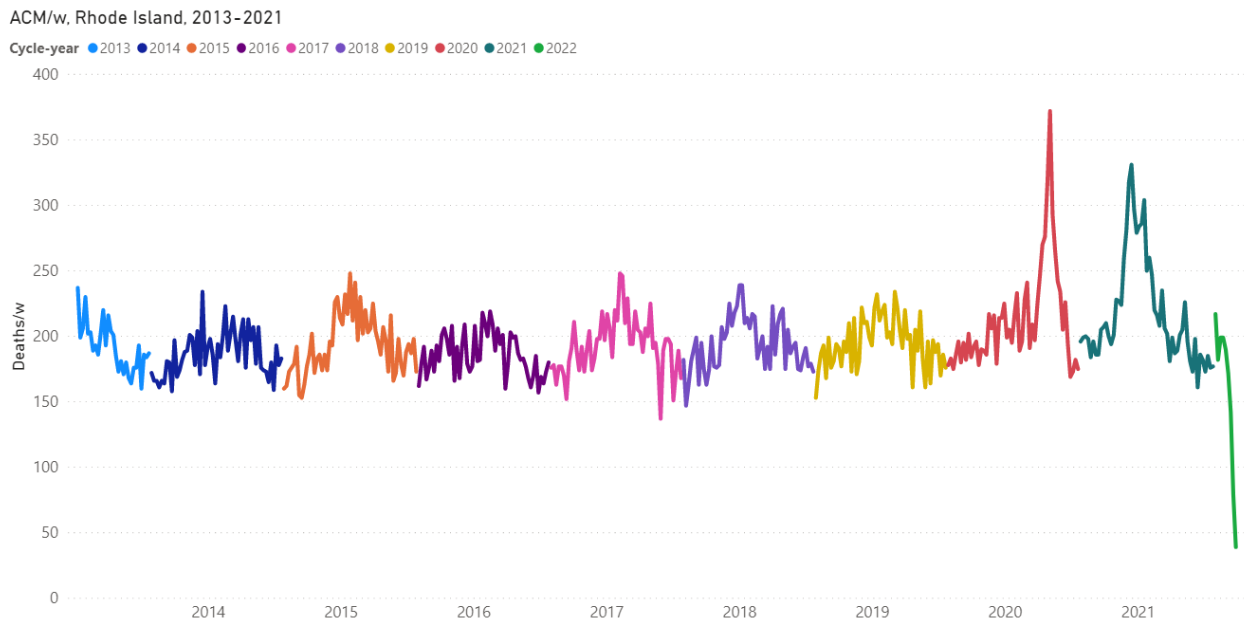


The last data point of Ohio is week-39 of 2021.

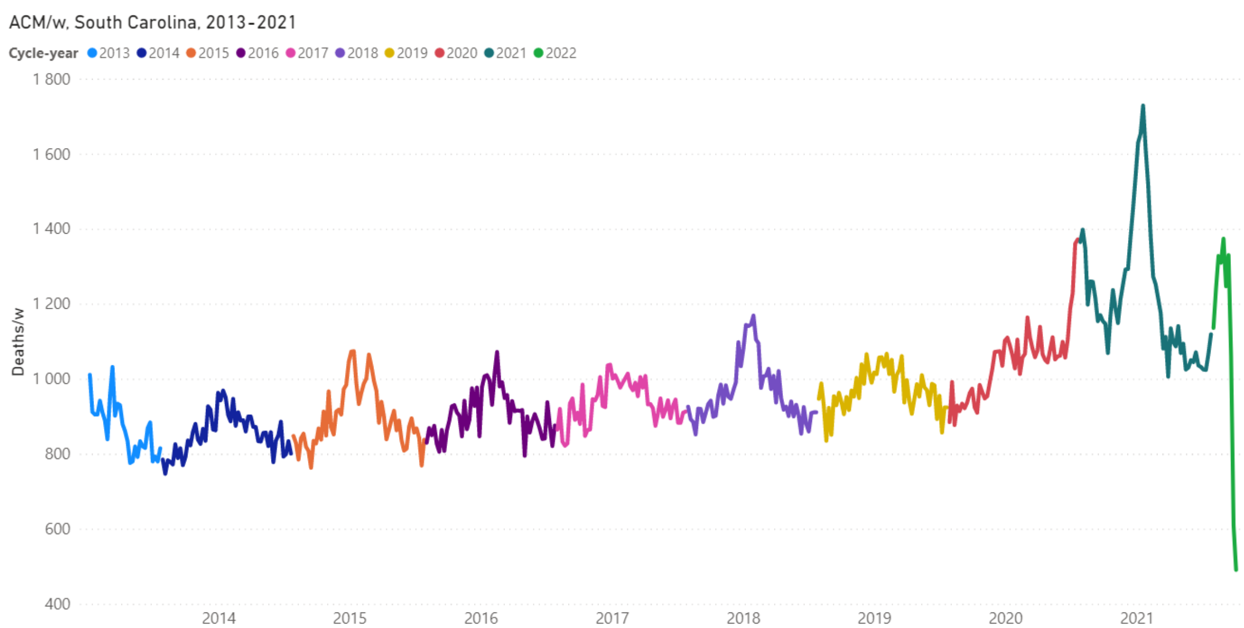


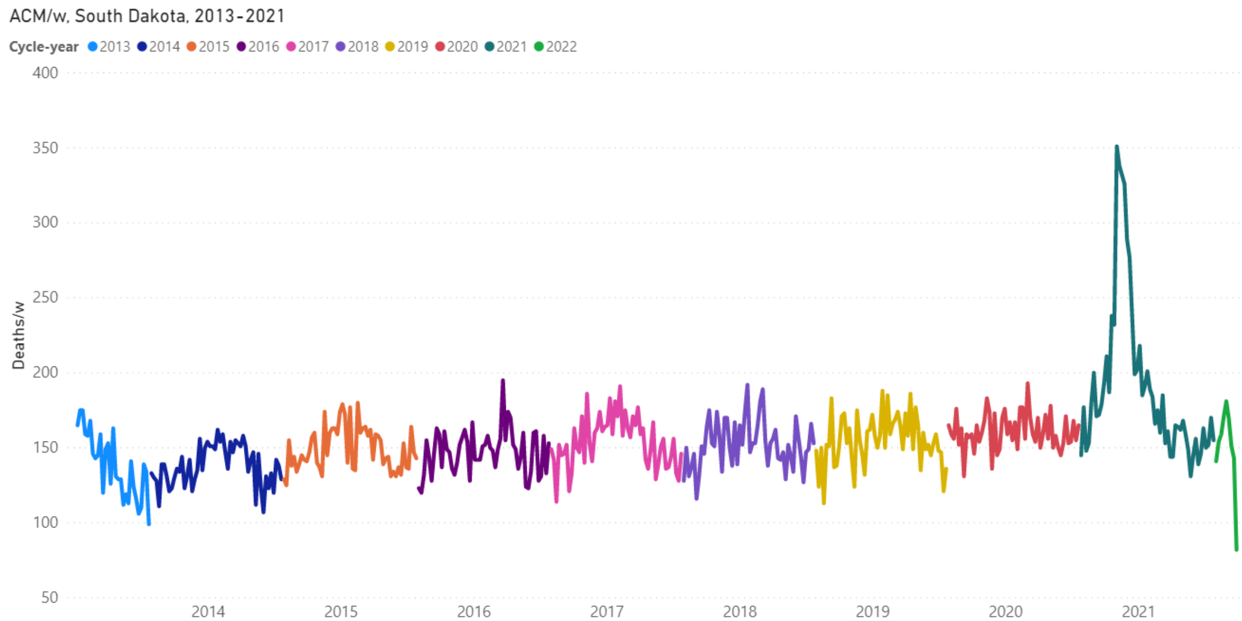

ACM/w, Oregon, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
Deaths/w
1 000
900
800
700
600
500
400
300
200
100
2014
2015
2016
2017
2018
2019
2020
2021


ACM/w, Pennsylvania, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
Deaths/w
4 500
4 000
3 500
3 000
2 500
2 000
1 500
1 000
2014
2015
2016
2017
2018
2019
2020
2021

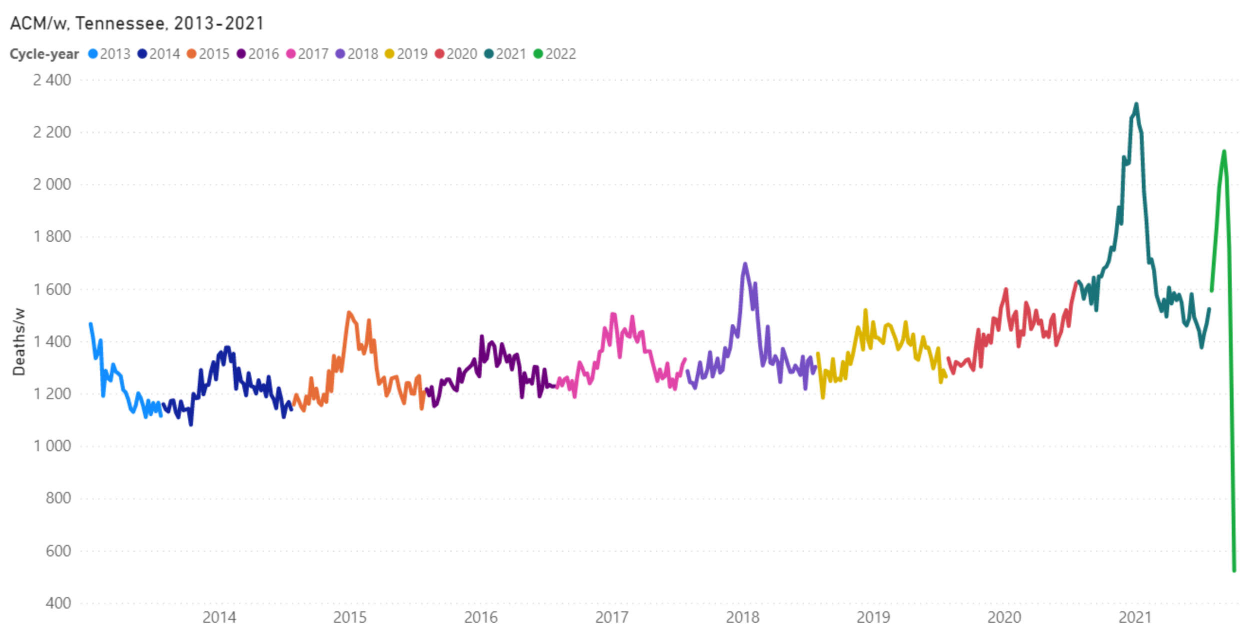


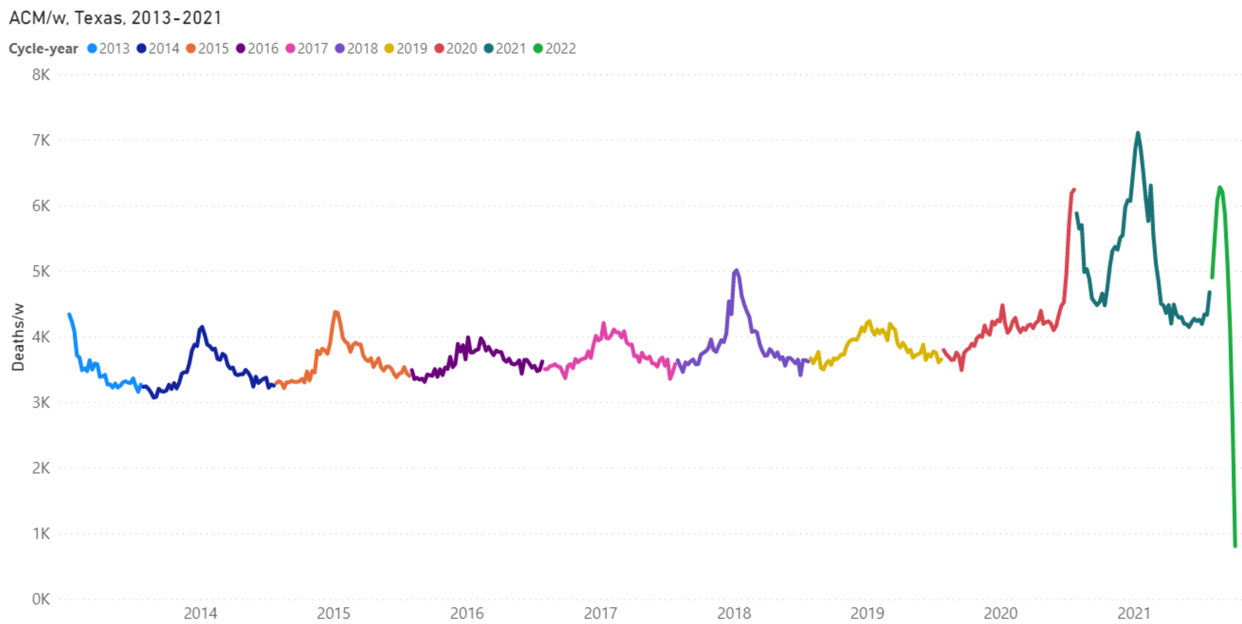
The last data point of Rhode Island is week-39 of 2021.

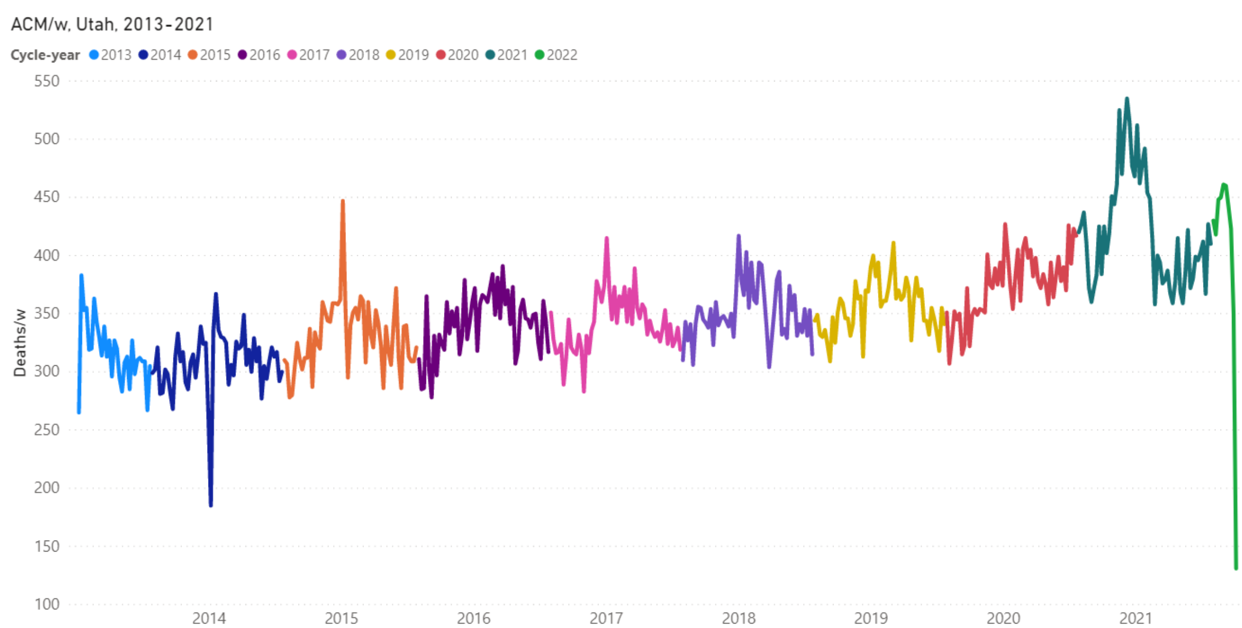


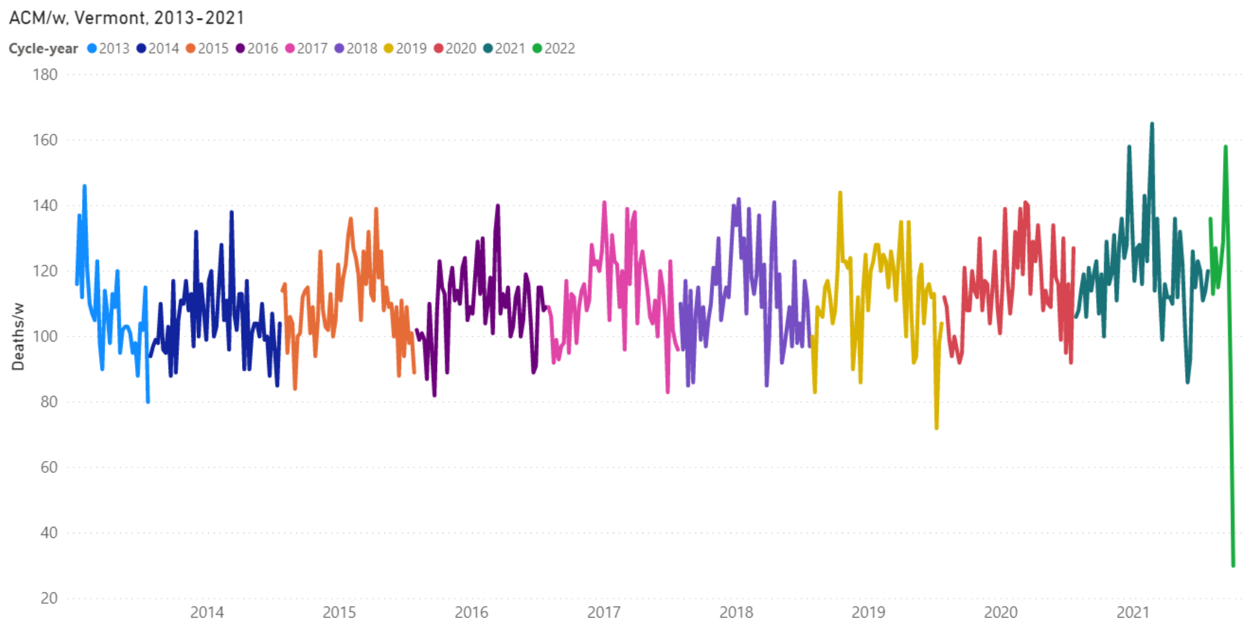


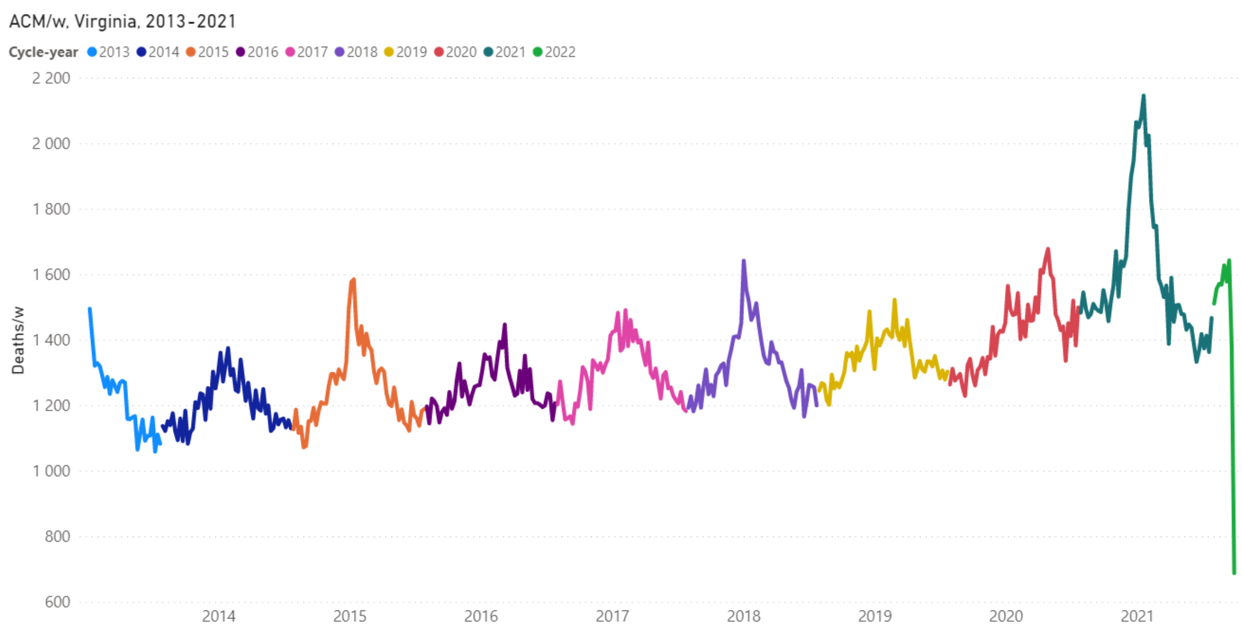
The last data point of South Dakota is week-39 of 2021.

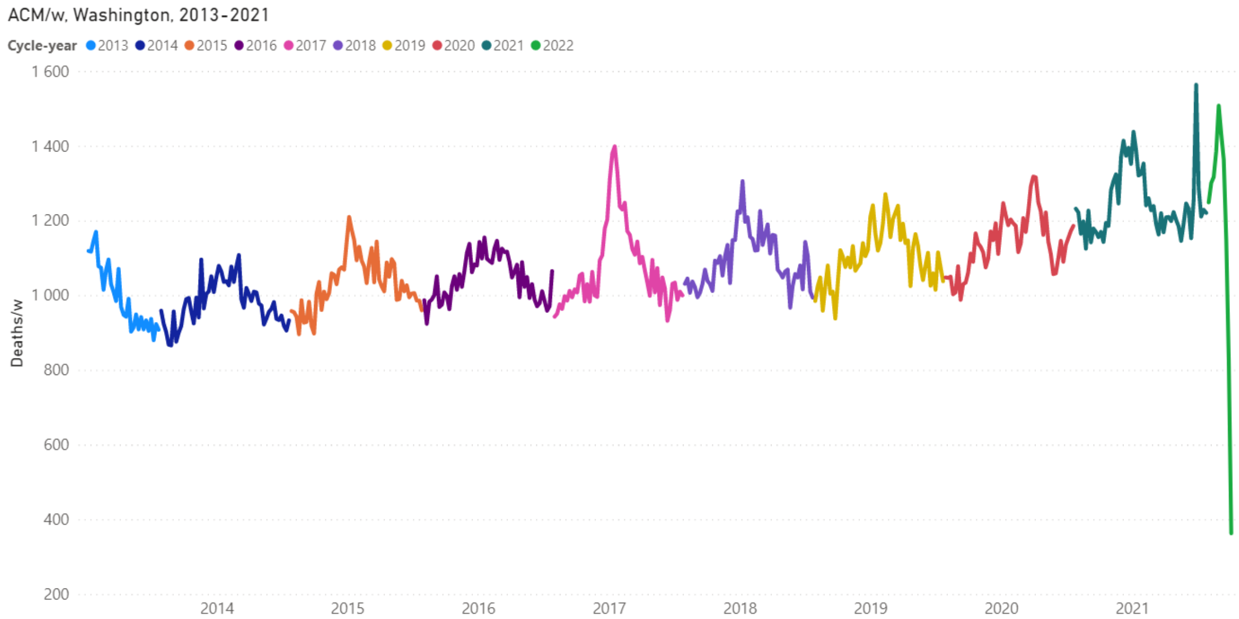


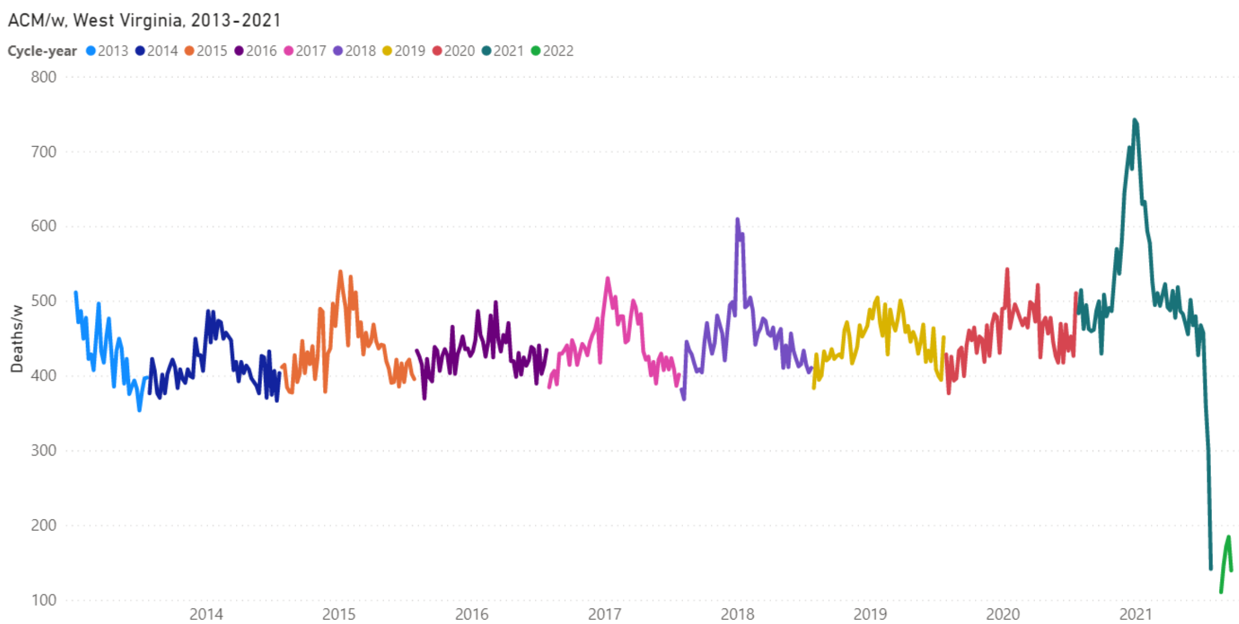

ACM/w, Texas, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
8K
7K
6K
5K
4K
3K
2K
1K
0K
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021


ACM/w, Utah, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
550
500
450
400
350
300
250
200
150
100
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021

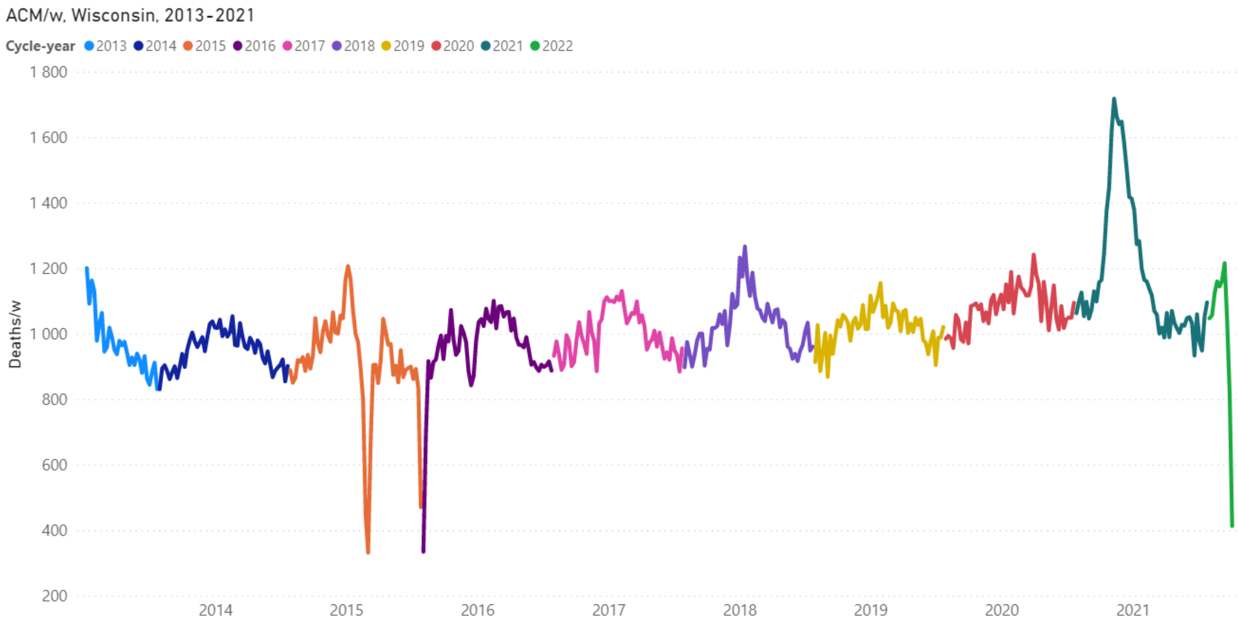

ACM/w, Vermont, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
180
160
140
120
100
80
60
40
20
Deaths/w
2014 2015 2016 2017 2018 2019 2020 2021

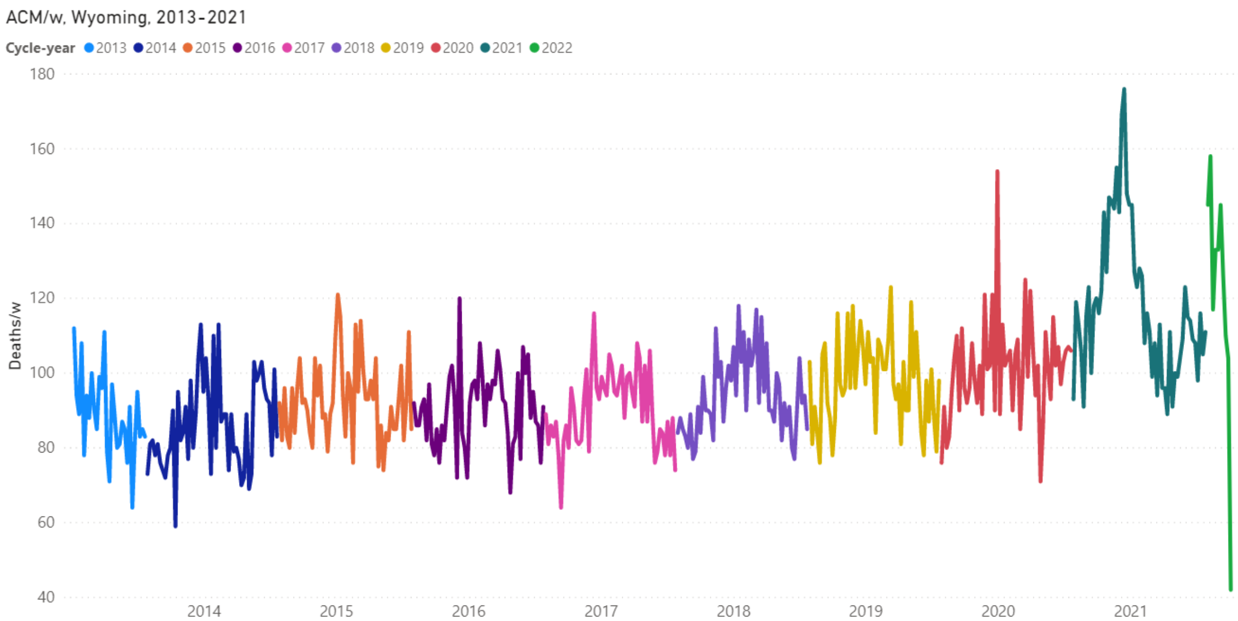

ACM/w, Virginia, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
2 200
2 000
1 800
1 600
1 400
1 200
1 000
800
600
Deaths/w
2014 2015 2016 2017 2018 2019 2020 2021





The last data point of West Virginia is week-38 of 2021.


ACM/w, Wisconsin, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
1 800
1 600
1 400
1 200
1 000
800
600
400
200
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021


ACM/w, Wyoming, 2013-2021
Cycle-year 2013 2014 2015 2016 2017 2018 2019 2020 2021 2022
180
160
140
120
100
80
60
40
Deaths/w
2014
2015
2016
2017
2018
2019
2020
2021

GlobalResearch
Center for Research on Globalization

Do Face Masks Reduce COVID-19 Spread in Bangladesh? Are the Abaluck et al. Results Reliable?

By Prof Denis Rancourt
Global Research, September 21, 2021

Region: Asia
Theme: Science and Medicine

***

Purpose

"This really should be the end of the debate," says Ashley Styczynski, an infectious-disease researcher at Stanford University in California and a co-author of the preprint describing the trial. The research "takes things a step further in terms of scientific rigour", says Deepak Bhatt, a medical researcher at Harvard Medical School in Boston, Massachusetts, who has published research on masking. — Nature | News | 09 September 2021 | "Face masks for COVID pass their largest test yet"

The leading trend-setting mainstream media and institutional public relations offices have been unreservedly enthusiastic about "the Bangladesh mask study" (see Appendix A).

Here, I review the methods and results of that study by Abaluck *et al.* (2021) published as a working paper by Innovations for Poverty Action (IPA): "The Impact of Community Masking on COVID-19: A Cluster-Randomized Trial in Bangladesh", 01 September 2021.

The study's stated primary outcome regarding the benefits of face masks is "symptomatic SARS-CoV-2 seroprevalence", meaning the prevalence during the study period of individuals self-reporting COVID-like symptoms who also test positive using a laboratory blood test presumed to be specific for SARS-CoV-2.

Summary

The cluster-randomized trial study of Abaluck *et al.* (2021) is fatally flawed, and therefore of no value for informing public health policy, for two main reasons:

1. The antibody detection was performed using a single commercial FDA emergency-use-authorized (EUA) serology test that is not suitable for the intended application to SARS-CoV-2 in Bangladesh (not calibrated or validated for populations in Bangladesh; undetermined cross-reactivity against broad-array IgM antibodies, malaria, influenza, etc.).

2. The participants (individual level, family level, village level) in the control and treatment arms were systematically handled in palpably different ways that are linked to factors established to be strongly associated to infection and severity with viral respiratory diseases, in particular, and to individual health in general.

These disjunctive fatal flaws are explained below. Either one is sufficient to invalidate the results and conclusions of Abaluck *et al.*

Furthermore, the Abaluck *et al.* symptomatic seroprevalence (SSP) results are *prima facie* statistically untenable. The treatment-to-control differences in numbers of symptomatic seropositive individuals are too small to rule out large unknown co-factor, baseline heterogeneity, and study-design bias effects. In addition, they are at best borderline significant, in terms of purely ideal-statistical estimations of uncertainty. Finally, the practice of using whole households while reporting on an individual basis, introduces unknown correlations/ clustering, and vitiates the mathematic assumptions that underlie the statistical method.

Can the chosen antibody test be used in this application?

Is the antibody assay specific for SARS-CoV-2?

A single laboratory test was used in the Abaluck *et al.* (2021) study: the “SCoV-2 Detect™ IgG ELISA” test kit (InBios, Seattle, Washington).

Here, ELISA stands for enzyme-linked immunosorbent assay, which is one of three main assay methods for routinely detecting or quantifying antibodies. IgG is a class of immunoglobulins. For the non-expert, two of the five classes of immunoglobulins, which are of relevance in the present critique, can be described as follows:

- Immunoglobulin M (IgM) – IgM antibodies are produced as a body’s first response to a new infection or to a new “non-self” antigen, providing short-term protection. They increase for several weeks and then decline as IgG production begins.
- Immunoglobulin G (IgG) – About 70-80% of the immunoglobulins in the blood are IgG. Specific IgG antibodies are produced during an initial infection or other antigen exposure, rising a few weeks after it begins, then decreasing and stabilizing. The body retains a catalog of IgG antibodies that can be rapidly reproduced whenever exposed to the same antigen. IgG antibodies form the basis of long-term protection against microorganisms. In those with a normal immune system, sufficient IgG is produced to prevent re-infection. Vaccinations use this process to prevent initial infections and add to the catalog of IgG antibodies, by exposing a person to a weakened, live microorganism or to an antigen that stimulates recognition of the microorganism. — *Merk Manuals* | Immunoglobulins (IgA, IgG, IgM) | accessed on 15 September 2021

Abaluck *et al.* (2021) state “This assay detects IgG antibodies against the spike protein subunit (S1) of SARS-CoV-2.” This statement is incorrect.

None of the official documents about the assay claim that the assay detects “the spike protein subunit (S1) of SARS-CoV-2”, or any part(s) of the spike protein. Rather, only a broad claim is ever made, of the type “The SCoV-2 Detect IgG ELISA is authorized for the detection

of antibodies to SARS-CoV-2 in human serum or plasma" or "INTENDED USE: The SCoV-2 Detect™ IgG ELISA is an in vitro diagnostic test for the qualitative detection of IgG antibodies to SARS-CoV-2 in human serum or plasma":

- IFU LBL-0113-03 (English) (Instructions for Use) (19 May 2021)
- Brochure COVE-G
- FDA EUA Letter of Authorization COVE-G (14 May 2021)
- Health Care Provider Fact Sheet COVE-G (14 May 2021)

These documents are also available on the FDA website.

The only mention of "spike", which I could find, is that the FDA webpage "EUA Authorized Serology Test Performance" ("Content current as of 18 August 2021", accessed on 14 September 2021) has the title of the section for this assay as:

InBios SCoV-2 Detect IgG ELISA

Developer: InBios
Test: SCoV-2 Detect IgG ELISA
Technology: ELISA
Target: Spike

The latter FDA (Test Performance, 2021) webpage provides the independent scientific assessment in the "Test Facts" that were used for FDA EUA approval as "NCI's Frederick National Laboratory for Cancer Research Evaluation Report" (dated 13 July 2021; accessed on 14 September 2021).

The said independent scientific assessment (FNLCR, 2021) is the reference document for evaluating the assay used by Abaluck *et al.* (2021). The FNLCR (2021) report makes it clear that not only was the assay not validated for detecting any specific SARS-CoV-2 IgG antibody, but it was also not validated for any ability to distinguish IgM and IgG:

> "The positive samples selected may not reflect the distribution of antibody levels in patient populations that would be evaluated by such a test. Because all samples are positive for both IgM and IgG, this evaluation cannot verify that tests intended to detect IgM and IgG antibodies separately detect these antibodies independently."

Given the nonspecificity of IgM — by its very nature as an initial broad-array immune response — this means that the assay may have a high potential for cross-reactivity with a large spectrum of infections or conditions.

The manufacturer of the assay (InBios) reports having made an in-house (not independent) evaluation of "Cross-Reactivity (Analytical Specificity)" and reports no cross-reactivity for several antibodies to other viral infections and autoantibodies, based on small numbers (n = 3-8) of unspecified reference samples, as (InBios, IFU LBL-0113-03, 2021):

Category	Number of samples tested	Number reactive
Anti-Influenza A/B	7	0
Anti-Hepatitis B	5	0
Anti-Hepatitis C	5	0
Anti-Nuclear Antibody	5	0
Rheumatoid Factor	18	3
Human Anti-Mouse Antibody	3	0
Anti-HIV	8	0
Anti-Respiratory Syncytial Virus	4	0
Normal Human Sera	180	0

Presumably, the reference samples were chosen to have specific IgG of the tested viral infections, and would therefore have little or no residual IgM initially induced by the tested infections, since IgG is generated as IgM decreases as functions of time from onset of symptoms.

From this Table (InBios, IFU LBL-0113-03, 2021), one might ask: Since cross-reactivity for rheumatoid factor was detected (3/18) by testing 18 samples, why were more samples not used for the other diseases (at least 18 samples, say)? After all, there is no lack of influenza standards, for example. Otherwise, with the small number of samples used, it is entirely possible to have missed large incidences of cross-reactivity.

As it stands, cross-reactivity is reported solely for “Rheumatoid Factor” (3/18) (InBios, IFU LBL-0113-03, 2021). Given this known cross-reactivity of the assay, Abaluck *et al.* should have obtained baseline prevalence of rheumatoid arthritis and Sjogren’s syndrome in their control and intervention arms, especially for their most elderly cohorts (50-60 and 60+ years) and for the two types of face masks, or they should have ruled out these conditions in their elderly “symptomatic seropositive” individuals, especially in view of their most surprising results (their Figure 3). Abaluck *et al.* did not do this (did not report doing this).

Yadouleton *et al.* (2021) studied cross-reactivity (specificity) of the InBios SCoV-2 Detect™ IgG ELISA assay, and of another ELISA assay nominally for SARS-CoV-2 antibodies. Of 60 pre-COVID (2019) samples from Benin, they found that the InBios assay gave many samples that were near the positive/negative threshold (“cut-off”) (their Figure 1A, fourth panel). They concluded, from the results for both assays: “acute malaria is the most plausible explanation for unspecific SARS-CoV-2 ELISA reactivity in prepandemic controls”, and found false positive rates as high as 25% (for the non-InBios assay).

The study of Yadouleton *et al.* (2021) is especially relevant because “Bangladesh is one of the four major malaria-endemic countries in South-East Asia having approximately 34% of its population at risk of malaria [...] with a prevalence ranging between 3.1% and 36%” (Islam *et al.*, 2013). Abaluck *et al.* did not report having surveyed or screened for past or present infections of malaria among their study subjects.

Is the antibody assay validated for use in Bangladesh?

The short answer is “no”. The long answer is as follows.

To start, we need accurate definitions of test specificity and sensitivity, which are provided, in the words of the FDA (Test Performance, 2021), as:

> The performance of these [EUA authorized serology] tests is described by their

> “sensitivity,” or their ability to identify those with antibodies to SARS-CoV-2 (true positive rate), and their “specificity,” or their ability to identify those without antibodies to SARS-CoV-2 (true negative rate).

There are two major problems with application of the InBios antibody assay to populations in Bangladesh.

The first major problem is that the performance of the emergency utilization authorized InBios test has never been evaluated for a real-world population; not in the USA, and not in Bangladesh. In the words of the independent evaluators (FNLCR, 2021) (p. 4):

> Samples used in this evaluation were not randomly selected, and sensitivity (PPA) and specificity (NPA) estimates in this report may not be indicative of the real-world performance of the InBios International Inc. SCoV-2 Detect™ IgG ELISA. [...]
>
> 1.3 Important caveats
>
> Sensitivity and specificity estimates in this report may not be indicative of the real world performance of the InBios International Inc. SCoV-2 Detect™ IgG ELISA. [...]
>
> The number of samples in the panel is a minimally viable sample size that still provides reasonable estimates and confidence intervals for test performance, and the samples used may not be representative of the antibody profile observed in patient populations.

The second major problem is as follows.

The InBios test is based on optical density (OD) measurements through the ELISA solution in the final step of the assay: the more reactive the sample (to the ELISA substrate intended to bind the target antibody), the greater the OD. The measured OD is divided by “the average OD plus three standard deviations” for many reference samples presumed to be free of the target antibody. This ratio ($OD_{sample}/OD_{cut\text{-}off}$), called the “Immunological Status Ratio” (ISR), is used to discriminate “positive” (ISR ≥ 1.1) and “negative” (ISR ≤ 0.9) samples. The manufacturer considers ISR values of >0.9 through >1.1 to be “borderline”/undetermined results.

In the words of the manufacturer (InBios, IFU LBL-0113-03, 2021) (p. 10):

> The assay cut-off value was determined by screening a large number (>100) of normal human serum (NHS) samples that were collected [in the USA] prior to the COVID-19 outbreak (~November, 2019). The cut-off selection was performed by estimating the mean of the negative specimens plus three (3) standard deviations.

Therefore, the determination of $OD_{cut\text{-}off}$ is critical and its value depends on the population from which one draws the so-called NHS samples. We can presume that InBios drew its NHS samples from a USA population, and that its arbitrary choices of “1.1/0.9 ISR thresholds” and “plus three (3) standard deviations” were made in order to “make it work”. That is, in order to resolve “positive” from “negative” serum samples, from USA residents known independently to test positive for SARS-CoV-2.

It is not reasonable to expect that the thus adopted test values ($OD_{cut\text{-}off}$, and 1.1/0.9 ISR thresholds) determined using “NHS” from USA residents would apply to a population of

Bangladesh citizens, because the pre-COVID "normal human serums" from Bangladesh citizens would be significantly different, regarding the prevalence of antibodies to various viral infections, autoantibodies, and cross-reactivity with immune-response products from various other infections (e.g., malaria) and conditions (e.g., rheumatoid arthritis, Sjogren's syndrome).

Indeed, even entirely within the USA, Kaufman *et al.* (2021), in their large study of "More than 2.4 million SARS-CoV-2 IgG serology (initiated April 21, 2020) and 6.6 million nucleic acid amplification testing (NAAT) (initiated March 9, 2020) results on persons from across the United States as of July 10, 2020", found that: "SARS-CoV-2 IgG positivity was observed in 91% (19,434/21,452) of individuals tested after a positive [nucleic acid amplification testing] NAAT result and in 10% (7,831/80,968) after a negative NAAT result. Factors associated with seropositivity include age, region of patient residence, and interval between NAAT and IgG serology."

To be clear, Kaufman *et al.* (2021) found that both the rate of IgG positivity among NAAT-positive individuals (~sensitivity) and the rate at which NAAT-negative individuals had subsequent IgG positivity (~false-positive rate) differed significantly with respect to geographic area within the USA: 93.4% to 86.2% and 16.4% to 4.8%, respectively, in going from the 5-state NE area (NY/NJ/MA/RI/CT) to all other states (their Figure 3).

Therefore, we must assume that there can be a large systematic difference in serology test performance and/or in population immunological response or characteristics in going from the USA to Bangladesh. The estimated magnitude of this systematic effect, indicated by the extensive results of Kaufman *et al.* (2021) for different geographical regions in the USA, is large enough to invalidate those results from Abaluck *et al.* that involve small differences in numbers of tested individuals, such as the impact of surgical masks on the most elderly cohorts, even if there were not the serious validation problems outlined above for the InBios test.

Furthermore, purely in terms of population immunology, do USA and Bangladesh populations have different prevalences, at any given time, of broad-array IgM, which the InBios test is not established to resolve from IgG?

Specifically, the spectrum of disease prevalence in Bangladesh is dramatically different than in the USA. Bangladesh has a "high" degree of risk (2020) for (The World Factbook): bacterial and protozoal diarrhea, hepatitis A and E, typhoid fever, dengue fever, malaria, leptospirosis, and rabies; and an obesity rate of 3.6 % (2016), compared to the USA obesity rate of 36.2% (2016) (adult prevalence rate).

Serum matrix effects ("cross-reactivity") must be expected to be large and different for Bangladesh, compared to the USA. Irrespective of anything else, or of any manufacturer's claims, Abaluck *et al.* (2021) should have stringently tested a representative array of known (independently and reliably determined) positive and negative serum samples from Bangladesh, using the InBios test as provided. Without this minimal precaution of upfront verification to rule out differences and to validate test utility, their test results are useless for the intended scientific purposes.

Was "spectrum bias" duly examined by InBios and Abaluck et al.? Are the positives reliable?

The answer is "no", at least on the basis of what is reported.

"Spectrum bias" is the unavoidable variation of performance of a test arising from the frequency distribution ("spectrum") of values that are being measured by the test in the given tested population (for example, see: Usher-Smith *et al.*, 2016).

Two problems occur.

1. At calibration: a test can have a significantly different actual performance than the performance evaluated using any set or array of known samples if the manufacturer's calibration (for setting of cut-off and undetermined range, and for assay protocol development) uses solely means and standard deviations, without regard to the shape of the distribution of test measurements (OD values) of the calibration samples (the ">100 of normal human serum (NHS)" samples used by InBios). This can produce misleading and over-enthusiastic test performance characteristics, and it again demonstrates the importance of using representative calibration samples.
2. In the field: a test can have significantly different performances (sensitivity, specificity) on different populations having different distributions of test measurements (OD values), even if the populations are otherwise comparable (comparable cross-reactive pathogens, co-factors, age structure, health status, etc.).

One simple consequence of the "spectrum bias" effect is that, in populations with low prevalence, many of the test results are close to the positive/negative threshold value, leading to particularly large errors, in general. This is why the FDA states (FDA, Test Performance, 2021) (p. 2):

> In low prevalence populations, the result of a single antibody test is not likely to be sufficiently accurate to make an informed decision regarding whether or not an individual has had a prior infection or truly has antibodies to the virus. A second test, typically one assessing for the presence of antibodies to a different viral protein, generally would be needed to increase the accuracy of the overall testing results.

This is also why the FDA (Test Performance, 2021) (p. 47) estimates a theoretical 95% confidence interval of (50.5%, 100%) in the positive predictive value (PPV) (probability of a positive being correct) for 5% population prevalence for the InBios test, despite the stellar EUA evaluation numbers.

This means that, depending on "prevalence" of the assay-reactive condition in the Bangladesh study populations of Abaluck *et al.*, the reliability of a positive determination can be 50% or less for small prevalence. Abaluck *et al.* report symptomatic prevalences of 0.76% (control arm) and 0.68% (intervention arm).

In the present case, the "test measurement" or "value that is being measured" is the above-described ratio ($OD_{sample}/OD_{cut\text{-}off}$), called the "Immunological Status Ratio" (ISR), obtained for a given serum sample using the InBios assay. It is a continuous variable, and it is obviously prone to "spectrum bias" since the manufacturer even defines an undetermined region, for ISR >0.9 through >1.1, rather than simply a definite positive/negative threshold value.

Therefore, if InBios wanted users and evaluators to gauge the potential for "spectrum bias", then it would, among other things, publish the distribution of ISR values of its large number of so-called normal human serum (NHS) samples that were collected in the USA prior to

COVID (InBios, IFU LBL-0113-03, 2021). I could not find such information, or any discussion of this issue. Likewise, the FNLCR (2021), in its evaluation of the test, discloses only positive/negative status, not ISR values for the evaluation samples.

Similarly, Abaluck *et al.* do not disclose their ISR values, do not show distributions of ISR values, and do not even state how many of their samples gave "undetermined" ("equivocal") ISR values on initial measurement (Abaluck *et al.*, 2021):

> [...] the immunological status ratio (ISR) was calculated as the ratio of optical density divided by the cut-off value. Samples were considered positive if the ISR value was determined to be at least 1.1. Samples with an ISR value 0.9 or below were considered negative. Samples with equivocal ISR values were retested in duplicate, and resulting ISR values were averaged.

For example, are the distributions of ISR values different for the control and intervention arms? We do not know.

Conclusion regarding the serology test

In conclusion, the FDA emergency-use-approved (EUA) InBios serology test was improperly applied by Abaluck *et al.* (2021):

1. It is not specific to SARS-CoV-2, since it has undetermined cross-reactivity against broad-array IgM antibodies (n=0), undetermined cross-reactivity with other corona viruses (n=0), probable cross-reactivity with malaria (peer-reviewed article), known cross-reactivity with rheumatoid factor (n=18), insufficiently tested cross-reactivity with influenza A/B (n=7), hepatitis B (n=5), hepatitis C (n=5), respiratory syncytial virus (n=4), and others, undetermined cross-reactivity (n=0) with the high-risk pathogens endemic to Bangladesh (bacterial and protozoal diarrhea, hepatitis A and E, typhoid fever, dengue fever, malaria, leptospirosis, and rabies), and unknown comparative serum matrix effects in USA and Bangladesh.
2. It has not been validated with any actual population, whether in the USA or Bangladesh, and is calibrated solely using USA serum samples.
3. It is not calibrated or validated for Bangladesh, and cannot be used as-given on residents of Bangladesh.

I find it unacceptable that a test that is not approved for patients —

> LIMITATIONS: ... • Assay results should be interpreted only in the context of other laboratory findings and the total clinical status of the patient. (InBios, IFU LBL-0113-03, 2021) (p. 12)

— would be used to diagnose participants in a trial, as having COVID-19, without any clinical evaluation beyond self-reporting of symptoms with survey questions, in order to justify long-term application of a treatment to millions of people, which has known and unknown associated harms (Rancourt. 2021).

Are the control and treatment arms valid (comparable)?

Let me start by stating the obvious, since it seems to have escaped detection by virtually all

media and public-relations reviewers (including the folks at *Nature*): A trial in which the researchers spend significant resources to convince the non-control group to accept or adopt the treatment is not a “randomized” trial, nor is it “controlled”. Rather, it is a trial in which one group is chosen to be intrusively manipulated to receive the treatment, whereas the other group is free from this manipulation. The trial design is not one in which the treatment and control groups are distinguished by the presence or absence of treatment, as the sole systematic difference. In addition, in this case, individuals in both groups are free to adopt the treatment or not, and that choice is anything but random, in both groups. If anything, the study of Abaluck *et al.* is in-effect merely another comparative study, but with extensive researcher interference.

Treatment alone versus adding super-treatment interventions

The study of Abaluck *et al.* (2021) suffers from a major difficulty: the researchers must apply significant and repeated interventions (in a campaign to induce acceptance of the treatment of mask wearing) to the treatment arm, while preventing those interventions in the treatment arm from inducing bias in the outcome.

In other words, the cluster-randomized study is worse than merely unblinded. It is a case in which the treated individuals are not solely subjected to the treatment (mask wearing), but are additionally subjected to the sustained and multi-faceted campaign of interventions to induce acceptance of the treatment.

It is one thing to design and evaluate interventions intended to generate mask use, but it is quite another thing to measure the health impact of increased mask use alone, without introducing co-factors arising from the interventions.

One way to reduce potential bias would have been to measure prevalence of the disease solely in families in the treatment arm (treatment villages) randomly selected not to be subjected to the interventions, if that were possible with redesigned interventions. However, this was not done. Prevalence in the treatment arm was measured in the same individuals and families that were subjected to the interventions.

This is not a fatal flaw if there are compelling and empirically supported reasons to believe that the additional (super-treatment) measures cannot affect the outcome. However, in this case, the opposite is true: there are compelling reasons to expect that the super-treatment measures affect the outcome, as explained below.

The basic super-treatment intervention consisted of the following elements, as described by Abaluck *et al.* (2021):

> To emphasize the importance of mask-wearing, we prepared a brief video of notable public figures discussing why, how, and when to wear a mask. The video was shown to each household during the mask distribution visit and featured the Honorable Prime Minister of Bangladesh Sheikh Hasina, the head of the Imam Training Academy, and the national cricket star Shakib Al Hasan. During the distribution visit, households also received a brochure based on WHO materials depicting proper mask-wearing.
>
> We implemented a basic set of interventions in all treatment villages, and cross-randomize additional intervention elements in randomly chosen subsets of treatment villages to investigate whether those have any additional impact on mask-wearing. The

basic intervention package consists of five main elements:

1. One-time mask distribution and promotion at households.
2. Mask distribution in markets on 3-6 days per week.
3. Mask distribution at mosques on three Fridays during the first four weeks of the intervention.
4. Mask promotion in public spaces and markets where non-mask wearers were encouraged to wear masks (weekly or biweekly).
5. Role-modeling and advocacy by local leaders, including imams discussing the importance of mask-wearing at Friday prayers using a scripted speech provided by the research team.

Participants, mask promoters, and mask surveillance staff were not blinded as intervention materials were clearly visible.

Science of the stress-immune relationship

The science background to understand why the interventions of Abaluck *et al.* would have an impact on prevalence is as follows.

First, researchers performing comparative trials for outcomes involving immune response must make themselves aware that ordinary psychological stress significantly impacts immune response, and that psychoneuroimmunology is a large field of research (Ader and Cohen, 1993).

Social status, within a specific dominance hierarchy, is a major predictor of chronic stress, in social animals including humans (Cohen *et al.*, 1997a) (Sapolsky, 2005), which, in turn, may be the dominant determinant of individual health, disease burden, and longevity (Cohen et al., 2007).

Ordinary psychological stress is known to be a dominant factor in making an individual susceptible to viral respiratory disease symptomatic infection, and to increase the severity of the infection (Cohen et al., 1991). Also, social isolation (paucity of social-network interactions), in addition to individual psychological stress, is known to have an added impact on the individual's susceptibility to viral respiratory disease (Cohen et al., 1997b).

Furthermore, there is a large age gradient: extended periods of psychological stress are known to have more deleterious health effects in elderly persons than in younger persons (Prenderville et al., 2015).

The stress-immune relationship, however, is not simply a monotonic function of integrated intensity. Frequency and duration are pivotal: chronic or long-term stress harms immune response, whereas short-term adaptive stress enhances immune response. The often-cited review by Dhabhar (2014) has:

> Short-term (i.e., lasting for minutes to hours) stress experienced during immune activation enhances innate/primary and adaptive/secondary immune responses. Mechanisms of immuno-enhancement include changes in dendritic cell, neutrophil, macrophage, and lymphocyte trafficking, maturation, and function as well as local and systemic production of cytokines. In contrast, long-term stress suppresses or dysregulates innate and adaptive immune responses by altering the Type 1–Type 2

> cytokine balance, inducing low-grade chronic inflammation, and suppressing numbers, trafficking, and function of immunoprotective cells.

Peters *et al.* (2021) have reviewed these concepts and the known science for the relevance to COVID-19. They pointed out that "the socioeconomic issues and various aspects of the Western type lifestyle that are closely associated with psychosocial stress have recently been reported to contribute to COVID-19". Their ultimate aim is to "clarify whether psychosocial interventions have the potential to optimize neuroendocrine-immune responses against respiratory viral infections during and beyond the COVID-19 pandemic."

Mechanisms of bias from the super-treatment interventions

Given the above-reviewed knowledge, it seems clear to me that Abaluck *et al.* (2021) have failed to consider a critical issue in their study design. Their interventions are interpersonal and societal interactions. All such interactions either induce or relieve psychological stress experienced by the individual, to different degrees and of different durations.

Specific elements (1 to 5) of the "basic intervention package" implemented by Abaluck *et al.* can be anticipated to modulate psychological stress in the following ways:

> (1) The distribution visit to each household in the treatment arm: "The video was shown to each household during the mask distribution visit and featured the Honorable Prime Minister of Bangladesh Sheikh Hasina, the head of the Imam Training Academy, and the national cricket star Shakib Al Hasan. During the distribution visit, households also received a brochure based on WHO materials depicting proper mask-wearing."
>
> Such a visit would provide (as it appears to have been intended to provide) hierarchical validation to the family members, thus raising the experienced social status, and reducing the dominance-hierarchy stress, experienced by lower strata, below its pre-visit long-term baseline value.
>
> (2, 3) The masks themselves would serve as a visual symbol of belonging to this thereby privileged group, and the regular mask distributions (in markets and at mosques) would be a constant interactive confirmation of an appreciative and caring hierarchical authority; all of which boosts the perceived increased social status, and reduces or displaces dominance-hierarchy stress.
>
> (4) "Mask promotion in public spaces and markets where non-mask wearers were encouraged to wear masks (weekly or biweekly)": "mask promoters patrolled public areas a few times a week and asked those not wearing masks to put on a mask." (Abaluck *et al.* found that excluding this element produced an increase in mask use of 10.9%, compared to 28.4% when it was included.)
>
> Such interactions are classic short-term, mostly unpredictable and repeated stress events, precisely of the type that "enhances innate/primary and adaptive/secondary immune responses" (Dhabhar, 2014).
>
> (5) "Role-modeling and advocacy by local leaders, including imams discussing the importance of mask-wearing at Friday prayers using a scripted speech provided by the research team"
>
> "Role-modeling" would again strengthen the perceived increased social status, and

reduce dominance-hierarchy stress. "Advocacy" can be oppressive, but it can also be of a more collaborative nature, which would work better when the advocate cannot surveil or enforce, and which would again work to reduce long-term dominance-hierarchy stress below the pre-study baseline.

Therefore, given what is known about stress-immune relations, the super-treatment interventions applied by Abaluck *et al.* would thereby enhance immune responses in the participants in the treatment arm, and consequently would reduce the probability of developing symptoms and of being infected, irrespective of any effect arising from filtration by the face masks.

Peters *et al.* (2021) envisage and argue for preventative treatment by stress management strategies precisely for COVID-19.

Furthermore, a successful socializing and educational campaign to the effect that face masks provide safety would be anticipated to create a bias towards a smaller tendency to recognize and report symptoms. In the Abaluck *et al.* study, symptoms were reported by phone or in person survey-interviews with the heads of families.

Thus, the trial design in the Abaluck *et al.* study has foreseeable built-in biases probably acting in the same direction. Their experimental design with interventions is fatally flawed, and the results are therefore of no value, irrespective of the problems with the blood test.

Is the size of the trial sufficient for the results to be reliable?

All adults, 18 through 60+ years old, both mask types together

There were approximately 170 K individuals in each arm of the study, which is a large number (Abaluck *et al.*, 2021). This does not in itself guarantee statistically reliable results, depending on the sizes of the cohort-specific treatment-to-control differences being reported, compared to the relevant theoretical standard deviations of the presumed purely ideal-statistical variations.

(I emphasize "ideal-statistical" because, as explained below, Abaluck *et al.* used households of closely interacting family members but then reported individual-based results, which vitiates the underlying theoretical assumptions of "independent, uncorrelated and random" in all the (ideal) statistical calculations of uncertainties and confidence intervals.)

From this sample size (170 K), there were approximately 13.5 K individuals in each arm who were reported to have developed "COVID-like symptoms" within the measurement time of the study: 13,273 (7.62%) (treatment), 13,893 (8.62%) (control). The control-treatment difference of 620, is significant since it is 5 times greater than the ideal-statistical standard deviations of the numbers prior to taking their difference, sqrt(13.5 K).

The numbers of symptomatic individuals having positive serology test results, and their treatment-control differences, however, are much smaller. Abaluck *et al.* (2021) chose not to report these numbers but instead reported only "symptomatic seroprevalence" (SSP), as percentages, after accounting for the rates (~40 %) of consent to the blood test (RCB): 0.68 % (treatment), 0.76 % (control).

I work backwards from their numbers to calculate the numbers of symptomatic individuals having positive blood test results, as follows:

Treatment arm:

178,288 participants x 0.0068 (SSP) x 0.408 (RCB) = 495 (2σ≈44) symptomatic seropositive individuals

→Scaled to the same population as the control → 455 (2σ≈41)

Control arm:

163,838 participants x 0.0076 (SSP) x 0.399 (RCB) = 497 (2σ≈45) symptomatic seropositive individuals

These formulas are correct if my contextual interpretation of the following (ambiguous) passage is correct: "Omitting symptomatic participants who did not consent to blood collection, symptomatic seroprevalence was 0.76% in control villages and 0.68% in the intervention villages. Because these numbers omit non-consenters, it is likely that the true rates of symptomatic seroprevalence are substantially higher (perhaps by 2.5 times, if non-consenters have similar seroprevalence to consenters)."

The difference, 497 – 495 = 2 individuals, is the number giving rise to Abaluck *et al.*'s difference in absolute symptomatic seroprevalence (SSP) of 0.0008. As such, given the expected sources of bias and measurement errors described herein, and given the size of this difference of only two (2) events, the SSP difference on increased masking in the treatment arm, reported by Abaluck *et al.*, cannot be taken as anything but unreliable.

The difference of "2 individuals" is 10 times smaller than the approximate ideal-statistical standard deviations (1σ) of the numbers prior to taking their difference, for comparable size starting populations. This should give anyone pause.

If I pursue the calculation to obtain a prevalence ratio (PR), including 95 % confidence intervals,

PR = 455 [414, 496] ÷ 497 [452, 542] = 0.92 [0.80, 1.04],

which is not statistically different from 1, and which gives a false impression of being borderline significant, from the purely ideal-statistical perspective.

Abaluck *et al.* report their results as: "Adjusting for baseline covariates, the intervention reduced symptomatic seroprevalence by 9.3% (adjusted prevalence ratio (aPR) = 0.91 [0.82, 1.00]; control prevalence 0.76%; treatment prevalence 0.68%)."

In fact, their bold assertion of a relative reduction in SSP of "9.3%", without stating its ideal-statistical error, while ignoring all other-than-ideal-statistical errors, is a fiction.

It is also misleading for Abaluck *et al.* to present their percent relative reduction in SSP with two significant numbers (as "9.3%"): without "adjustment", I calculate a percent relative reduction in SSP ((497 – 455)/497) of 8.4 % ± 12.2 % (2σ), which is consistent with zero.

Oldest age group, 60+ years old, surgical masks only

In their most surprising result, Abaluck *et al.* (2021) report a statistically significant three-significant-digit "34.7 %" relative decrease in symptomatic seroprevalence (from 1.03 % to

0.69 %, from control to treatment) among the 60+ years old age cohort, for surgical masks only in the treatment arm (their Figure 3).

Among other reasons, this result is surprising because all the many (>10) policy-grade randomized controlled trials (RCT) with lab-verified outcomes, for COVID-19 and other viral respiratory diseases, have found no statistically significant benefit from either surgical or N95 masks, in terms of transmission and infection. I have reviewed this context here: (Rancourt, 2021) (Rancourt, 2020a) (Rancourt, 2020b) (Rancourt, 2020c).

It is difficult to evaluate the said most surprising result of Abaluck *et al.* because the authors do not provide:

- the numbers of 60+ year olds in each group (control vs treatment with surgical masks)
- the fraction of distributed surgical masks to all distributed masks, in treatment-arm 60+ year olds
- the numbers of symptomatic 60+ year olds in each group (control vs treatment with surgical masks)
- the rate of consent to the blood test (RCB) in each group (control vs treatment with surgical masks)

On 13 September 2021, I emailed Dr. Abaluck directly and asked for these and other numbers of individuals: "… Basically, I am asking to know these 30 most basic numbers, only a few of which are already provided in your article. Can you or one of your co-authors provide these?" Dr. Abaluck responded the same day, as: "We will be posting replication instructions publicly in a few weeks and you'll be able to see all the data. If you can't find it in 3 weeks or so, please feel free to reach out again."

I note that Abaluck *et al.* (2021) do not provide ideal-statistical error estimates (confidence intervals) for any of their symptomatic seroprevalence numbers, for any group or arm. This leaves me with an impression of avoiding reporting estimated statistical uncertainties; while dealing solely with group to group differences and group to group relative changes of seroprevalence values having unreported error estimations.

Without the numbers for the 60+ year olds, it is impossible to definitively verify ideal-statistical uncertainty in the said most surprising result. Nonetheless, the needed uncertainties can be estimated using what is provided, by making reasonable assumptions for the missing information, as follows.

For this purpose: I assume the same RCB for 60+ year olds (control, surgical masks) as for all adults in the same arm. I assume that 16 % of adults in all groups are 60+ year olds (The World Factbook, for Bangladesh, 2020). I assume that 66.7 % of 60+ year olds receiving masks received surgical masks, equal to the cross-randomization fraction on a village basis (200/300).

I then estimate the numbers of symptomatic 60+ year olds having positive blood test results, as follows:

Treatment group, 60+ year olds, surgical masks:

178,288 participants x 0.16 (fraction 60+) x 0.667 (faction surgical masks) x 0.0069

(SSP) x 0.408 (RCB)

= 54 (2σ≈15) symptomatic seropositive 60+ year olds, surgical masks

→Scaled to the same population as the control → 74 (2σ≈21)

Control group, 60+ year olds:

163,838 participants x 0.16 (fraction 60+) x 0.0103 (SSP) x 0.399 (RCB)

= 108 (2σ≈21) symptomatic seropositive 60+ year olds, control

Thus I estimate that the two comparable numbers of symptomatic seropositive 60+ year old individuals overlap within their 95 % confidence intervals (74 [53, 95] (treatment); 108 [87, 129] (control)), from purely ideal-statistical considerations.

As a check, my numbers give a prevalence ratio (PR), 60+ year olds, surgical masks:

PR = 74 [53, 95] (treatment) ÷ 108 [87, 129] (control) = 0.69 [0.45, 0.92],

which is close to the "adjusted" PR reported by Abaluck *et al.*:

aPR = 0.65 [0.46, 0.85].

Whereas this PR (aPR) for 60+ year olds and surgical masks has an appearance of being mathematically valid, it is not reliable, for the following reasons:

1. The confidence interval is from purely ideal-statistical considerations. It is from the counting uncertainties alone, under ideal applicability assumptions. The main mathematical assumption is that each event or detection (of symptomatic seropositivity) is independent and random.
2. The actual (here estimated) absolute numbers of events or detections are small (54 and 108) and are therefore all the more susceptible to large errors from all sources, not just purely ideal-statistical counting errors. The smaller the cohorts, the greater the chance of contamination by unknown "baseline" factors, and the harder it is to secure a "balanced" comparison.
3. Observational bias error in reporting symptoms is expected, as explained above (impression of higher safety, unblind observers).
4. There is a built-in bias for resilience against infection in the treatment group, as explained above, which is expected to be strong, and is predicted to be strongest in the most elderly (stress-immune relation).
5. There is an insufficiently large blood-testing rate of consent (RCB, ~40 %), such that the non-randomized consent itself is therefore susceptible to bias.
6. The laboratory test is not specific to SARS-CoV-2, is not validated for Bangladesh, and is susceptible to large occurrences of "undetermined" or "equivocal" readings, as explained above, all of which make it susceptible to bias in whatever it is detecting or not detecting.
7. Many factors may be highly imbalanced between the treatment and control arms, which are not known or controlled in the study. These factors include infections, conditions or pathologies that have possible or likely cross-reactivity in the serology test, as explained above. This potential is probably higher in the most elderly, who are often afflicted with several co-conditions.

8. There is a large (50 %) imbalance in “baseline symptomatic seroprevalence rate”: 0.00002 (treatment), 0.00003 (control) (their “Table 1: Balance Tests (Individual-Level)” and “Table A3: Balance Tests (Village-Level)”). Abaluck *et al.* do not explain “rate” or discuss or attempt to interpret this apparently fundamental difference. This imbalance may indicate different immune histories or different immune health of the individuals or different pathogenic environments in the control and treatment arms.
9. There may be unaccounted or unknown correlations or clustering that vitiate the assumption of ideal-statistical independence and randomness. For example, a 60+ year old may have a higher-than-otherwise (higher than random) probability of being symptomatic seropositive if another 60+ year old in the same household is or recently was symptomatic seropositive, and so on. After all, the study includes all adults per participating household, rather than the common/standard study design of having independent participants. (This means that the method of calculation of confidence intervals for this study design, looking at individuals, is itself strictly invalid; as are all individual-base prevalence and prevalence-ratio results.)
10. There may be hidden co-factors that produce COVID-like symptoms and give cross-reactivity in the serology test. The door is wide open for this possibility since the COVID-19 symptoms are rather generic and the serology test is far from having been evaluated to be specific for SARS-CoV-2, as show above. The small absolute numbers of events or detections (54 and 108) allow such co-factors (one or several) to be accidentally different to a large extent in the two groups.
11. Symptomatic seropositivity for COVID-19 was not confirmed by clinical diagnosis; and symptomatic seroprevalence (SSP) was not validated by hospitalization data or mortality or prescription data or absenteeism, etc. Abaluck *et al.* give no information about number and severity of symptoms, but instead use a binary threshold of “symptomatic”. What was comparative symptomatology (severity, etc.) in the small numbers for the two groups (54 and 108)?

Conclusion

The Abaluck *et al.* (2021) study is an extreme case in which a Bayesian analysis of the impact of foreseeable potential bias and measurement uncertainty would confirm that their results are false, but the sophisticated demonstration is hardly necessary (Ioannidis, 2005) (Greenland, 2006).

In technical language, it is a case of “garbage in, garbage out”, not to mention the fundamental design flaws including using households while extracting individual-base results, and applying impactful super-treatment interventions to the treatment arm.

If this is the new “gold-standard clinical trial” (according to *Nature*) then the value of gold has plummeted to that of lead.

And see: Appendix A.

*

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internet forums. etc.

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Denis G. Rancourt, PhD is a Researcher at Ontario Civil Liberties Association (ocla.ca).

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Appendix A: Media reviews of the Abaluck *et al.* (2021) mask study

A few features made me suspicious of the Abaluck *et al.* (2021) study. The first was the high octane media campaign, followed by my noting the presence of clearly false statements in the media articles.

Another was the self-serving and incomplete description of the context of face mask efficacy studies, made by the authors themselves, in-effect ignoring all existing policy-grade trials that find no detectable advantage to mask wearing, in terms of transmission and infection. Abaluck *et al.* summarise as: "Inspired by the growing body of scientific evidence that face masks can slow the spread of the disease and save lives [refs], we conducted…"; and they never attempt to reconcile their surprising results with the existing science.

I infer that Abaluck *et al.* may self-justify in-effect ignoring all past work by distinguishing "source control" and "protective effect" of face masks? They sate: "First, unlike technologies with primarily private benefits, mask adoption is likely to yield especially large benefits at the community-level." This concept of "the one-way mask" is not based of any empirical evidence of actual person-to-person transmission. It also seems contrary to mechanistic expectations. If masks filter relevant particles, then they should filter them in both directions, both inhaling and exhaling. Exhaling is towards the outside environment, whereas inhaling is directly towards the respiratory tract tissue that is the target of the pathogen. If face masks are "one-way" then it should be the other way.

Here is a sample of the media reports:

— *Nature* | News | 09 September 2021 | "Face masks for COVID pass their largest test yet"

> Face masks protect against COVID-19. That's the conclusion of a gold-standard clinical trial in Bangladesh, which backs up the findings of hundreds of previous observational and laboratory studies.[ref].
>
> Critics of mask mandates have cited the lack of relevant randomized clinical trials, which assign participants at random to either a control group or an intervention group. But the latest finding is based on a randomized trial involving nearly 350,000 people across rural Bangladesh. The study's authors found that surgical masks — but not cloth masks — reduced transmission of SARS-CoV-2 in villages where the research team distributed face masks and promoted their use.
>
> "This really should be the end of the debate," says Ashley Styczynski, an infectious-disease researcher at Stanford University in California and a co-author of the preprint describing the trial. The research "takes things a step further in terms of scientific rigour", says Deepak Bhatt, a medical researcher at Harvard Medical School in Boston, Massachusetts, who has published research on masking. …

— *Stanford Medicine* | News Center | 01 September 2021 | "Surgical masks reduce COVID-19 spread, large-scale study shows"

> The findings were released Sept. 1 on the Innovations for Poverty Action website, prior to their publication in a scientific journal, because the information is considered of pressing importance for public health as the pandemic worsens in many parts of the world.
>
> "We now have evidence from a randomized, controlled trial that mask promotion increases the use of face coverings and prevents the spread of COVID-19," said Stephen Luby, MD, professor of medicine at Stanford. "This is the gold standard for evaluating public health interventions. Importantly, this approach was designed be scalable in lower- and middle-income countries struggling to get or distribute vaccines against the virus."

— *The Washington Post* | 01 September 2021 | "Massive randomized study is proof that surgical masks limit coronavirus spread, authors say"

> The authors of a study based on an enormous randomized research project in Bangladesh say their results offer the best evidence yet that widespread wearing of surgical masks can limit the spread of the coronavirus in communities.
>
> The preprint paper, which tracked more than 340,000 adults across 600 villages in rural Bangladesh, is by far the largest randomized study on the effectiveness of masks at limiting the spread of coronavirus infections.
>
> Its authors say this provides conclusive, real-world evidence for what laboratory work and other research already strongly suggest: mask-wearing can have a significant impact on limiting the spread of symptomatic covid-19, the disease caused by the virus.
>
> "I think this should basically end any scientific debate about whether masks can be effective in combating covid at the population level," Jason Abaluck, an economist at Yale who helped lead the study, said in an interview, calling it "a nail in the coffin" of the arguments against masks.

— *NBC News* | 01 September 2021 | "Largest study of masks yet details their importance in fighting Covid-19"

> A study involving more than 340,000 people in Bangladesh offers some of the strongest real-world evidence yet that mask use can help communities slow the spread of Covid-19.
>
> The research, conducted across 600 villages in rural Bangladesh, is the largest randomized trial to demonstrate the effectiveness of surgical masks, in particular, to curb transmission of the coronavirus. Though previous, smaller studies in laboratories and hospitals have shown that masks can help prevent the spread of Covid, the new findings demonstrate that efficacy in the real world — and on an enormous scale.
>
> "This is really solid data that combines the control of a lab study with real-life actions of people in the world to see if we can get people to wear masks, and if the masks work," said Laura Kwong, an assistant professor of environmental health sciences at the University of California, Berkeley, and one of the co-authors of the study.

— *Berkeley Public Health* | 01 September 2021 (undated) | "Largest study of its kind finds face masks reduce COVID-19"

> Wearing face masks, particularly surgical masks, is truly effective in reducing the spread of COVID-19 in community settings, finds a new study led by researchers from Yale University, Stanford Medical School, the University of California, Berkeley, and the nonprofit Innovations for Poverty Action (IPA). …
>
> "These results suggest that we could prevent unnecessary death and disease if we get people to wear high-performance masks, such as surgical masks, in schools, workplaces, shopping centers, places of worship and other indoor spaces," said study co-author Laura Kwong, an assistant professor of environmental health sciences at Berkeley's School of Public Health.

— *The Atlantic* | 04 September 2021 | "The Masks Were Working All Along"

> Now we have definitive proof that masks really are effective.
>
> … Their conclusion? Masks work, period. Surgical masks are particularly effective at preventing coronavirus transmission. And community-wide mask wearing is excellent at protecting older people, who are at much higher risk of severe illness from COVID-19.

— *Yale Daily News* | 13 September 2021 | "First randomized trial on masking affirms efficacy, Yale study says"

> … The 300,000-person study was the first randomized trial on mask efficacy.
>
> Yale professors of economics Ahmed Mushfiq Mobarak and Jason Abaluck, alongside a team of researchers from Stanford University and the University of California at Berkeley, conducted a cluster-randomized trial in rural Bangladesh that tested the intervention of community-level masking promotion from November 2020 to April 2021. …
>
> "A lot of conversation around mask usage previously had been that there had never been a randomized, controlled trial that demonstrated that masks were effective in both interrupting and preventing disease," said Stephen Luby, professor of infectious diseases at Stanford University and a coauthor of the study. "This really was a gold standard trial and was able to demonstrate just that."

— *WebMD Health News* | 07 September 2021 | "Large Study Confirms Masks Work to Limit COVID-19 Spread"

> The study demonstrates the power of careful investigation and offers a host of lessons about mask wearing that will be important worldwide. …
>
> "What we really were able to achieve is to demonstrate that masks are effective against COVID-19, even under a rigorous and systematic evaluation that was done in the throes of the pandemic," said Ashley Styczynski, MD, who was an infectious disease fellow at Stanford University when she collaborated on the study with other colleagues at Stanford, Yale, and Innovations for Poverty Action (IPA), a large research and policy nonprofit organization that currently works in 22 countries.

My competence to review science about COVID-19

I am a former tenured Full Professor of Physics, University of Ottawa, Canada. Full Professor is the highest academic rank. During my 23-year career as a university professor, I developed new courses and taught over 2000 university students, at all levels, and in three different faculties (Science,

Engineering, Arts). I supervised more than 80 junior research terms or degrees at all levels from post-doctoral fellow to graduate students to NSERC undergraduate researchers. I headed an internationally recognized interdisciplinary research laboratory, and attracted significant research funding for two decades.

I have been an invited plenary, keynote, or special session speaker at major scientific conferences some 40 times. I have published over 100 research papers in leading peer-reviewed scientific journals, in the areas of physics, chemistry, geology, bio-geochemistry, measurement science, soil science, and environmental science.

My scientific h-index impact factor is 41, and my articles have been cited more than 5,000 times in peer-reviewed scientific journals (profile at Google Scholar).

My personal knowledge and ability to evaluate the facts in this article are grounded in my education, research, training and experience, as follows (see this):

1. *Regarding environmental nanoparticles.* Viral respiratory diseases are transmitted by the smallest size-fraction of virion-laden aerosol particles, which are reactive environmental nanoparticles. Therefore, the chemical and physical stabilities and transport properties of these aerosol particles are the foundation of the dominant contagion mechanism through air. My extensive work on reactive environmental nanoparticles is internationally recognized, and includes: precipitation and growth, surface reactivity, agglomeration, surface charging, phase transformation, settling and sedimentation, and reactive dissolution. In addition, I have taught the relevant fluid dynamics (air is a compressible fluid), and gravitational settling at the university level, and I have done industrial-application research on the technology of filtration (face masks are filters).
2. *Regarding molecular science, molecular dynamics, and surface complexation.* I am an expert in molecular structures, reactions, and dynamics, including molecular complexation to biotic and abiotic surfaces. These processes are the basis of viral attachment, antigen attachment, molecular replication, attachment to mask fibers, particle charging, loss and growth in aerosol particles, and all such phenomena involved in viral transmission and infection, and in protection measures. I taught quantum mechanics at the advanced university level for many years, which is the fundamental theory of atoms, molecules and substances; and in my published research I developed X-ray diffraction theory and methodology for characterizing small material particles.
3. *Regarding statistical analysis methods.* Statistical analysis of scientific studies, including robust error propagation analysis and robust estimates of bias, sets the limit of what reliably can be inferred from any observational study, including randomized controlled trials in medicine, and including field measurements during epidemics. I am an expert in error analysis and statistical analysis of complex data, at the research level in many areas of science. Statistical analysis methods are the basis of medical research.
4. *Regarding mathematical modelling.* Much of epidemiology is based on mathematical models of disease transmission and evolution in the population. I have research-level knowledge and experience with predictive and exploratory mathematical models and simulation methods. I have expert knowledge related to parameter uncertainties and parameter dependencies in such models. I have made extensive simulations of epidemiological dynamics, using standard compartmental models (SIR, MSIR) and new models.
5. *Regarding measurement methods.* In science there are five main categories of measurement methods: (1) spectroscopy (including nuclear, electronic and vibrational spectroscopies), (2) imaging (including optical and electron microscopies, and resonance

imaging), (3) diffraction (including X-ray and neutron diffractions, used to elaborate molecular, defect and magnetic structures), (4) transport measurements (including reaction rates, energy transfers, and conductivities), and (5) physical property measurements (including specific density, thermal capacities, stress response, material fatigue…). I have taught these measurement methods in an interdisciplinary graduate course that I developed and gave to graduate (M.Sc. and Ph.D.) students of physics, biology, chemistry, geology, and engineering for many years. I have made fundamental discoveries and advances in areas of spectroscopy, diffraction, magnetometry, and microscopy, which have been published in leading scientific journals and presented at international conferences. I know measurement science, the basis of all sciences, at the highest level.

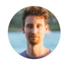
Analysis of all-cause mortality by week in Canada 2010- 2021, by province, age and sex: There was no COVID-19 pandemic, and there is strong evidence of response- caused deaths in t...

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Marine Baudin



Jérémie Mercier
Santé Liberté

Analysis of all-cause mortality by week in Canada 2010-2021, by province, age and sex: There was no COVID-19 pandemic, and there is strong evidence of response-caused deaths in the most elderly and in young males

Denis G. Rancourt[1,*], Marine Baudin[2], Jérémie Mercier[2]

[1] Ontario Civil Liberties Association (ocla.ca) ; [2] Mercier Production (jeremie-mercier.com) ;
* denis.rancourt@alumni.utoronto.ca

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6 August 2021

Abstract

We analyzed all-cause mortality by week (ACM/w) for Canada, and for the Canadian provinces, and by age group and sex, from January 2010 through March 2021; in comparison with data for other countries and their regions or counties.

We find that there is no extraordinary surge in yearly or seasonal mortality in Canada, which can be ascribed to a COVID-19 pandemic; and that several prominent features in the ACM/w in the COVID-19 period exhibit anomalous province-to-province heterogeneity that is irreconcilable with the known behaviour of epidemics of viral respiratory diseases (VRDs). We conclude that a pandemic did not occur.

In addition, our analysis of the ACM/w, by province, age and sex, allows us to highlight anomalies, occurring during the COVID-19 period, which provide strong evidence that:

- Among the most elderly (85+ years), many died from the immediate response to the pandemic that was announced by the WHO on 11 March 2020.
- Predominantly young males (0-44 years, and also 45-64 years) probably indirectly died from the sustained pandemic response, in the summer months of 2020, and into the fall and winter, starting in May 2020, especially in Alberta, significantly in Ontario and British Columbia, whereas not in Quebec.

Our study provides constraints on the mechanisms at play in VRD epidemics.

Index

1. Introduction

A viral respiratory disease (VRD) pandemic has two defining characteristics (Doshi 2008, 2011):

1. It occurs everywhere, irrespective of state or jurisdictional boundaries, presumably because there is no prior immunity.
2. It causes excess mortality far greater than that due to non-pandemic (seasonal) VRD epidemics.

In 2008, Doshi (2008) put it this way:

> One recent official US death toll projection(ref) suggested that the next pandemic will kill 6 to 56 times more Americans than the CDC currently estimates die in an average nonpandemic influenza season.(ref) The World Health Organization (WHO), in a “relatively conservative estimate,”(ref) predicted that the next influenza pandemic could claim 4 to 30 times more lives worldwide than a typical nonpandemic season.(ref)

One problem, in practice, is that VRD-classed mortality is difficult to quantify. The actual number of VRD-attributable deaths is always uncertain, especially when the deaths are counted in the context of a media-frenzy about “the pandemic”. This is as true today as it was when epidemiology was a nascent science; because a cause of death determination, with many co-factors, and in the absence of an analytical autopsy, is prone to human error, human bias, institutional bias, and even constructed bias as we have seen in the COVID period (Borger et al., 2021).

One solution is to avoid the problem altogether, by studying all-cause mortality (ACM) rather than VRD-classed mortality. A death is a death is a death.

In particular, if there is no discernable excess ACM during the presumed pandemic, above the trend in ACM, of the prior decade, say, then it is incorrect to conclude that a pandemic occurred.

The only alternatives are:

1. to believe that a pandemic occurred but that an extraordinary medical response prevented the presumably new pathogen from killing many people, in just the right amount as to bring the yearly ACM back to the decadal trend value; or
2. to believe that a pandemic occurred but that an extraordinary public-health response delayed the presumably new pathogen in its killing, in just the right amount as to bring the yearly ACM back to the decadal trend value, and then prevented future killing by an extraordinary mass vaccination campaign;

or some combination of the two, or their equivalents.

In science, there is a guiding principle regarding competing interpretations of the same data, called “Occam’s razor” (Gibbs, 1996):

> The most useful statement of the principle for scientists is: "when you have two competing theories that make exactly the same predictions, the simpler one is the better."

In this article, we ask whether a COVID-19 pandemic occurred in Canada, using the above criteria. Our application of Occam's razor, in this context, is supported by a multitude of studies showing that public-health measures are ineffective against a VRD, which we have reviewed in several other articles.[1]

2. Data

Statistics Canada (StatCan) is the national statistical office of the country. The all-cause mortality (ACM) data used in this article was retrieved from this database and is given by week (ACM/w) and covers the 2010-2021 period (StatCan, 2021). At the date of access, data were available from week-1 of 2010 (beginning of January) through week-17 of 2021 (end of April). In this article we present the data until week-12 of 2021 (end of March) because for later weeks the data for Canada are not consolidated and have the artifact of anomalously small mortality values.

The StatCan data are provided by:

- Provinces and territories
- Age group

[1] See: "COVID" section, Denis Rancourt's website: https://denisrancourt.ca/categories.php?id=1&name=covid (accessed on 5 August 2021).

- 0-44 years-old
- 45-64 years-old
- 65-84 years-old
- 85 years-old and over

- Sex
  - Males
  - Females

StatCan specifies that the ACM for 2020 and 2021 is provisional, and that the counts of deaths “have been rounded to a neighbouring multiple of 5 to meet the confidentiality requirements of the Statistics Act”.

3. Results / Interpretation

3.1 No detectable pandemic increase in the yearly and seasonal mortality

The all-cause mortality by week (ACM/w) for Canada, from January 2010 through March 2021, is shown in Figure 1a:



Figure 1a: All-cause mortality by week in Canada from 2010 to 2021. Data are displayed from January 2010 to March 2021. The y-scale is adjusted to show the region of interest. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

It is important to keep in mind that such graphs are represented using a region-of-interest y-scale. The same data on the full (starting at zero) y-scale is shown in Figure 1b:

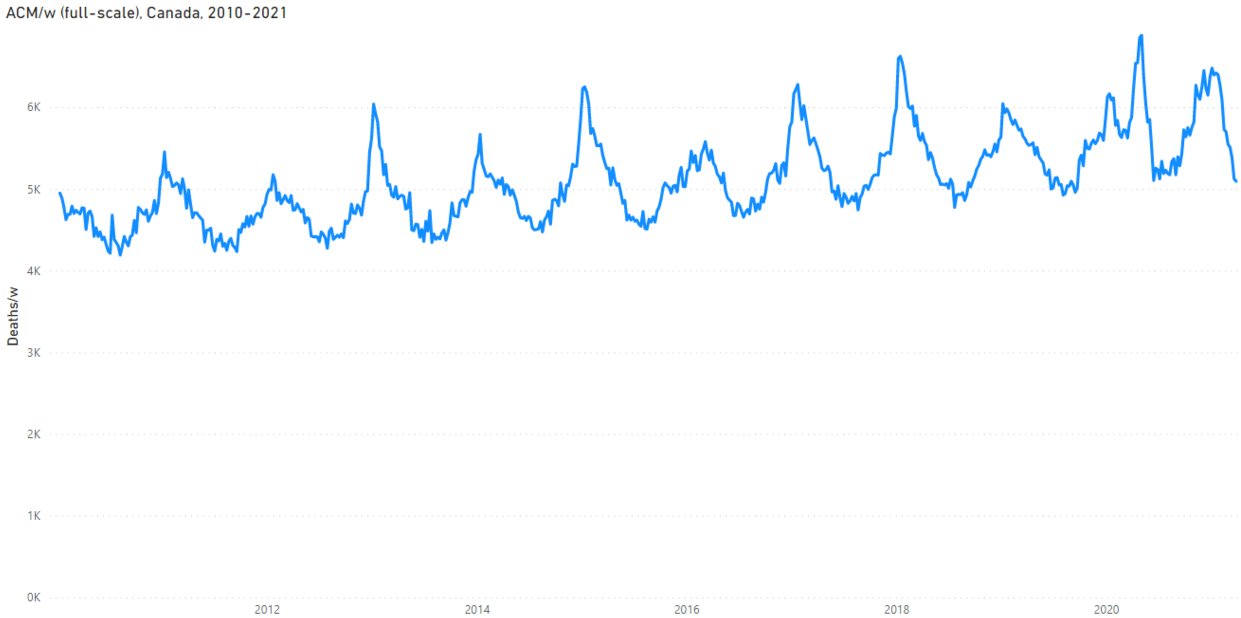


Figure 1b: All-cause mortality by week in Canada from 2010 to 2021. Data are displayed from January 2010 to March 2021. The y-scale is not adjusted to show only the region of interest; it starts from 0. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

In terms of the coarse-level main features (not intra-seasonal details), the usual seasonal pattern occurred in Canada since 2010 into March 2021, which is normally observed in all mid-latitude Northern hemisphere countries or jurisdictions, since 1900 or so where data has been collected.

The said usual seasonal pattern has these main features:

- winter highs and summer lows (here, of deaths per week, ACM/w)
    - summer-low or trough values (deaths per week) that vary monotonically from summer to summer, typically linearly over the course of a decade (we refer to this monotonic variation as the “summer baseline trend”)

    - winter-high or maximum values (deaths per week) that vary erratically from winter season to winter season, in both magnitude and date (or week-number)
- winter-burden deaths (integrated above the summer baseline trend, over a "cycle-year", from mid-summer to mid-summer) typically (since the 1960s) corresponding to between 5% and 15% of yearly mortality

We have analysed such patterns in ACM by time (day, week, month) for several jurisdictions, including jurisdictions in Canada, in two prior articles (Rancourt, 2020) (Rancourt, Baudin, Mercier, 2020).

Figure 1 shows that there was no excess yearly or seasonal mortality, above the usual values of the last decade for Canada, in either the 2019-2020 winter or the 2020-2021 winter (up to and including March 2021). This is confirmed by calculating ACM per year. We calculated ACM by "cycle-year", where we define a cycle-year as occurring from week-31 (around the beginning of August) of calendar year N through to week-30 (around the end of July) of calendar year N+1. As such, for example, nominal cycle-year 2018 is centered on the winter of 2018-2019. This definition of cycle-year takes one from mid-summer-trough to the next mid-summer-trough in ACM/w, such as to capture the intrinsic seasonal structure of ACM/w, having winter highs and summer lows. The result is plotted in Figure 2:

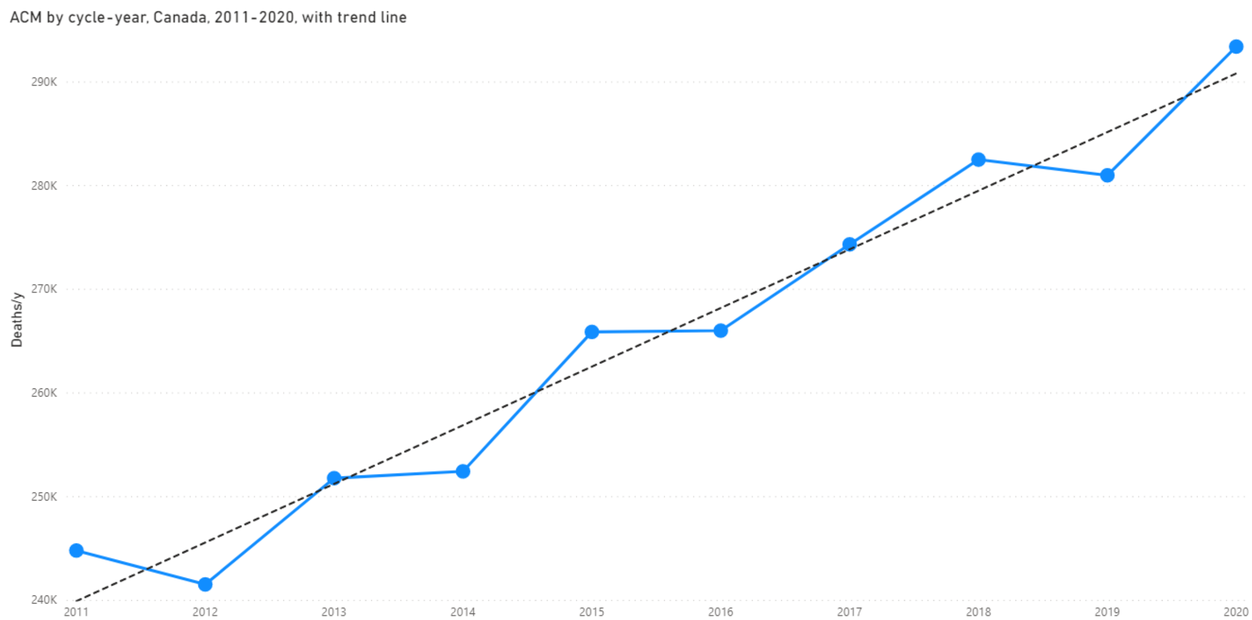


Figure 2: All-cause mortality by cycle-year for Canada, cycle-years 2011 to 2020, calculated as described above. The dashed line is a least-squares fitted straight line. The cycle-year starts on week-31 of a calendar year (beginning of August) and ends on week-30 of the next calendar year (end of July). Data for the calculation were retrieved from StatCan (StatCan, 2021), as described in section 2.

We conclude that there was no COVID-19 pandemic in Canada. It would be difficult to conclude otherwise. Either a pandemic causes a significant increase in deaths, or there was not a pandemic, barring the many unscientific false beliefs in effective public health interventions for VRDs.

Let us make this point further by showing the anomalous province-to-province intra-seasonal variations in ACM by time, which occur in the COVID or nominal-pandemic period (after 11 March 2020, the date the WHO proclaimed a pandemic).

3.2 Inter-jurisdictional uniformity of pre-COVID-period features in all-cause mortality by time, 2010-2019

The ACM/w 2010-2021 (through to March 2021) is plotted for several Canadian provinces, as follows.

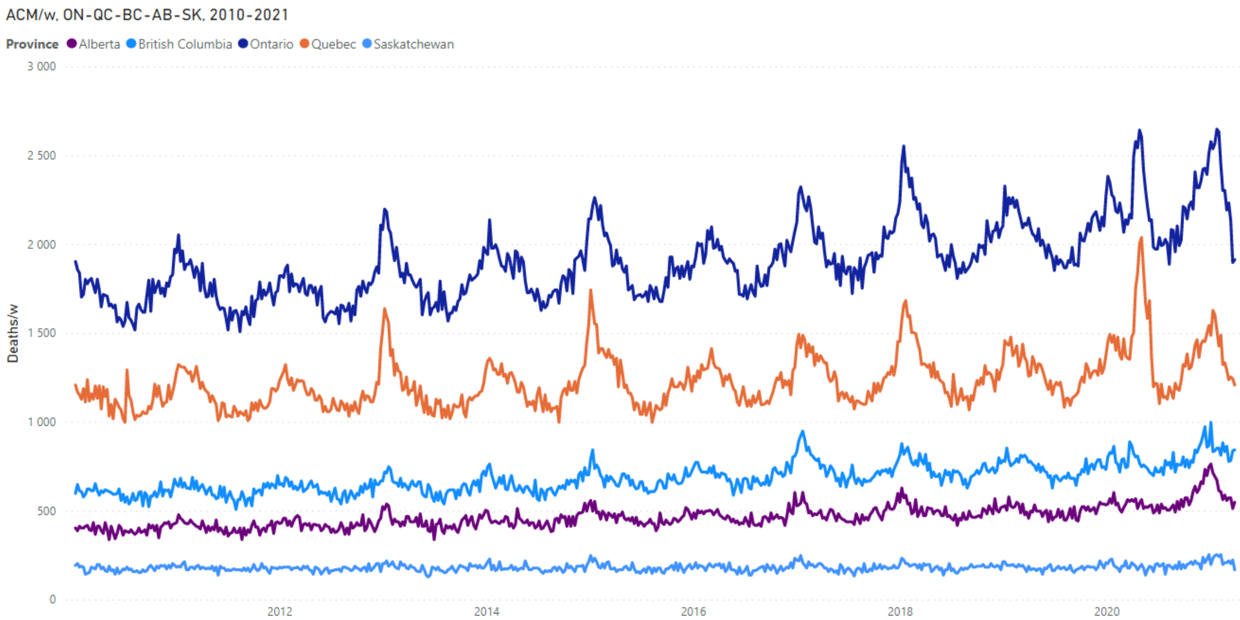


Figure 3a: All-cause mortality by week from 2010 to 2021 for, top to bottom, Ontario (ON), Quebec (QC), British Columbia (BC), Alberta (AB) and Saskatchewan (SK). Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

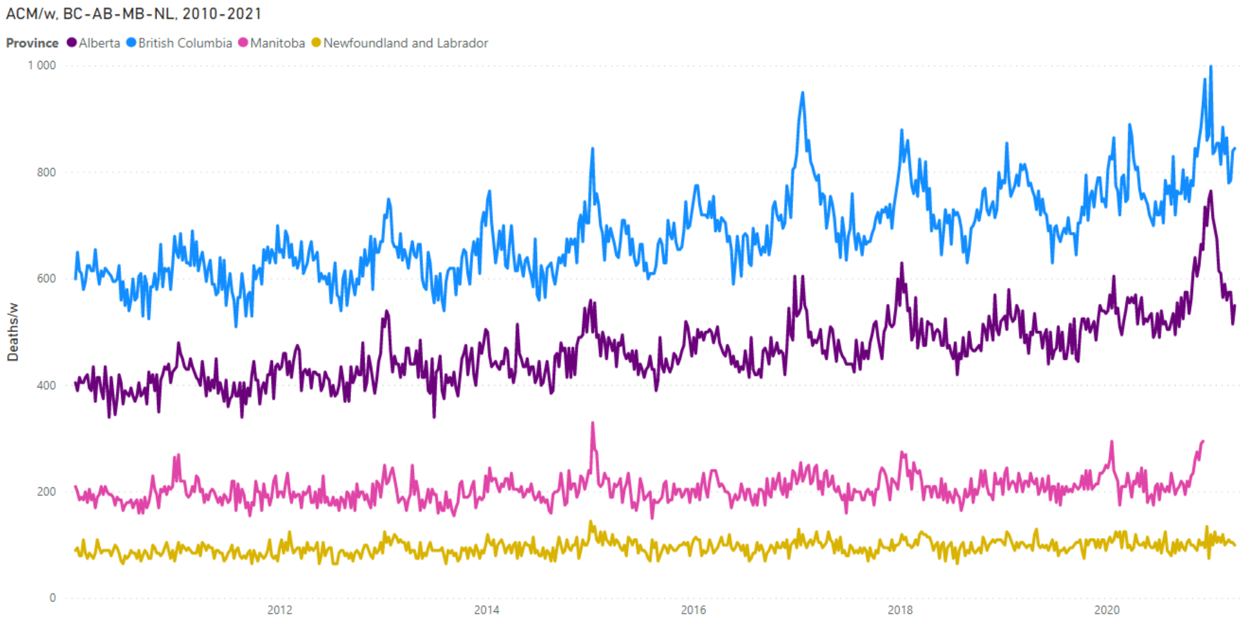


Figure 3b: All-cause mortality by week from 2010 to 2021 for, top to bottom, British Columbia (BC), Alberta (AB), Manitoba (MB) and Newfoundland and Labrador (NL). Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

The seasonal cycles of ACM/w are extraordinarily regular and display essentially identical winter-season features from province to province for a given winter, up to and including 2019. In other words, up to and including 2019, the seasonal patterns and intra-seasonal shapes of ACM/w are synchronous copies of each other, from province to province, while being scaled in whole-pattern magnitude approximately by provincial population. Plots of ACM/w, normalized by provincial population, are shown and compared in the Appendix.

We have observed such regularity, from jurisdiction to jurisdiction, and including from continent to continent, in all ACM-by-time data that we have examined for many jurisdictions (countries, regions, provinces, counties) in North America and Europe, over

the many decades of available data, for example (Rancourt, 2020) (Rancourt, Baudin, Mercier, 2020). Although there are small differences, the main first-level observation is the remarkable similarity in patterns, ratios of winter-to-winter magnitudes, and synchronicity, across all mid-latitude jurisdictions. We note that these robust data (ACM-by-time for North America and Europe, 20th and 21st centuries up to 2019) put into question two paradigms about VRDs (presumed to be the major cause of the seasonality of mortality in mid-latitude countries):

- that a specific VRD-causing virus/variant originates at a localized source and "spreads" across countries or continents by person to person contact or personal proximity ("source-spread" paradigm)
- that there are "pandemics" of VRDs, distinct from non-pandemic epidemics ("pandemic" paradigm)

Regarding the latter point, none of the 1957-1958 H2N2, 1968 H3N2, 2009 H1N1, or 2003 SARS pandemics are detected in ACM-by-time data, as meaningfully distinguished from non-pandemic seasonal epidemics. This is also the case if one analyses estimates of "influenza-classed mortality" rather than ACM (Doshi, 2008). The 1918 surges in ACM in both continents, by contrast, are very large, but constitute a special case involving mass bacterial infections, prior to the advent of antibiotics, killing solely young adults and infants, not the elderly, in societies and economies dramatically reorganized after the end of the First World War.

At the very least, ACM-by-time data imposes stringent real-world constraints on the theoretical or interpretational consequences of using these paradigms (source-spread, pandemic) to explain large-scale epidemiological observations.

Clearly for Canada, which is the size of a continent, Figures 3a & 3b (and see Appendix) show a remarkable regularity up to and including 2019: The provinces, East to West, have the same "fingerprints" of ACM/w. Detailed winter-season shapes, timing of features (synchronicity), and ratios of winter-to-winter magnitudes, are all essentially the same, province to province, 2010-2019, although the amplitudes of seasonal variation are smaller in the low-altitude (non-mountainous) maritime-climate provinces of the Canadian East coast (see below).

3.3 Inter-jurisdictional variations of COVID-period features in all-cause mortality by time

Although, as described above in section 3.1, "in terms of the coarse-level main features (not intra-seasonal details), the usual seasonal pattern occurred in Canada since 2010 into March 2021" (including the COVID-period), nonetheless there were significant anomalies in intra-seasonal features in the COVID-period, which we next examine, and which are relevant to whether a pandemic occurred.

As stated in the Introduction (section 1), a pandemic “occurs everywhere, irrespective of state or jurisdictional boundaries, presumably because there is no prior immunity”.

In particular:

- The pathogen presumed to cause the pandemic — a highly contagious pathogen of the VRD kind — will not stop at provincial borders in Canada.
- The presumed pathogen will not affect the similar populations in different provinces in dramatically different ways; such as killing young males in one province while killing only the elderly in another.
- The presumed pathogen itself, acting at the same time in March-April-May 2020 in two neighbouring similar provinces, for instance Ontario and Quebec, cannot be 2-3 times more deadly (per inhabitant) in Quebec than in Ontario.

We examine these propositions in the following figures.

First, the ACM/w for Canada is represented in an expanded view, from 2019 through March 2021, in order to define key features that occurred in the COVID-period:



Figure 4: All-cause mortality by week in Canada from 2019 to 2021. Data are displayed from January 2019 to March 2021. The dark-blue vertical line represents the week of March 11 2020, when WHO declared the pandemic. The three features are labelled as: C = "covid-peak", S = summer 2020, 2 = 2020-2021 winter peak ("2nd wave"). Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

Here, the 11 March 2020 date of the WHO pronouncement of the pandemic is shown as the vertical line, "C" denotes the ACM-by-time feature that we have called the "covid-peak" (Rancourt, 2020) (Rancourt, Baudin, Mercier, 2020), "S" denotes the summer trough in mortality of 2020, and "2" denotes the 2020-2021 winter peak (usually referred to as "2nd wave").

The Canada ACM/w features "C" and "S" (Figure 4) are anomalous in their own right, as follows.

We have already written extensively about “C”, which is our so-called “covid-peak”, observed in many jurisdictions in mid-latitude Northern hemisphere countries (Rancourt, 2020) (Rancourt, Baudin, Mercier, 2020). It is anomalous in that:

- Everywhere that it occurs, it emerges synchronously immediately following the WHO’s 11 March 2020 pronouncement of the pandemic.
- Its initial rise is exceedingly sharp, with a base to inflection-point time of approximately 3 weeks (2 weeks in ACM by day, ACM/d, data for France).
- Such a large and sudden surge virtually never occurs so late in the seasonal cycle (after 11 March, in March, April, May), which is otherwise always a downslope from the mid-winter (January-February) highs.
- It is extremely heterogeneous by jurisdiction in its magnitude, not being present or barely detected in 34 of the 52 USA states, 6 of the 13 regions of metropolitan France, 7 of the 10 provinces of Canada, 18 of the 21 counties of Sweden, and so on, while being disproportionately large in specific jurisdictions such as New York City in the USA, the Paris region in France, Stockholm county in Sweden, and the province of Quebec in Canada.
- Where it occurs, the degree to which it extends late into the season (into May) is variable from jurisdiction to jurisdiction; ending in April 2020 in France, in May 2020 in Canada and the USA.

The Canada ACM/w feature “S” (Figure 4) is anomalous because its mean baseline magnitude (5.25K deaths/w) is anomalously larger than the summer-2019 mean baseline value (5.05K deaths/w), and significantly larger than the magnitude predicted

by the linear summer baseline trend values for the prior years, as can be ascertained from Figure 1.

This means that some net 200 excess deaths per week were occurring in Canada in the summer of 2020, in a season in which VRDs are not active. Below, we show that the main contributor to these excess summer deaths was deaths of young (0-44 years) males, an age where COVID-19 virtually does not cause deaths (Levin et al., 2020), occurring predominantly in Alberta, Ontario and British Columbia. Whereas, the opposite occurs in Canada for the 85+ years age group: The summer-2020 mean baseline magnitude (ACM/w) is significantly smaller than the 2010-2019 trend value for this age group (Figure 6a).

Figures 3a & 3b show the following points regarding the COVID-period:

- Only ON, QC and BC have significant “C”-features (“covid-peaks”). The other seven provinces do not have statistically detectable “C”-features.
- The “C”-feature in the QC data is very strong, intermediate in ON, and relatively weak in BC.
- Whereas AB, MB and SK did not have “C”-features, they have anomalously large “2”-features, compared to their prior winter-season mortalities since 2010, especially AB.

These observations are easier to make in y-scale expanded views of each province:



Figure 5-ON: All-cause mortality by week in Ontario from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

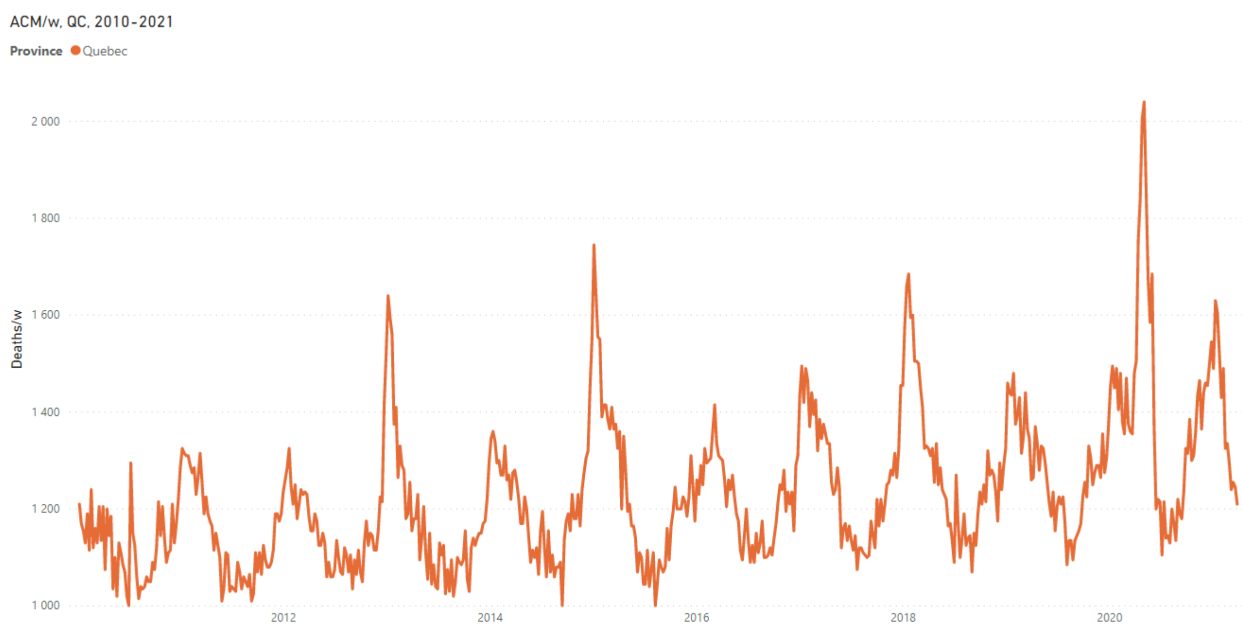


Figure 5-QC: All-cause mortality by week in Quebec from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

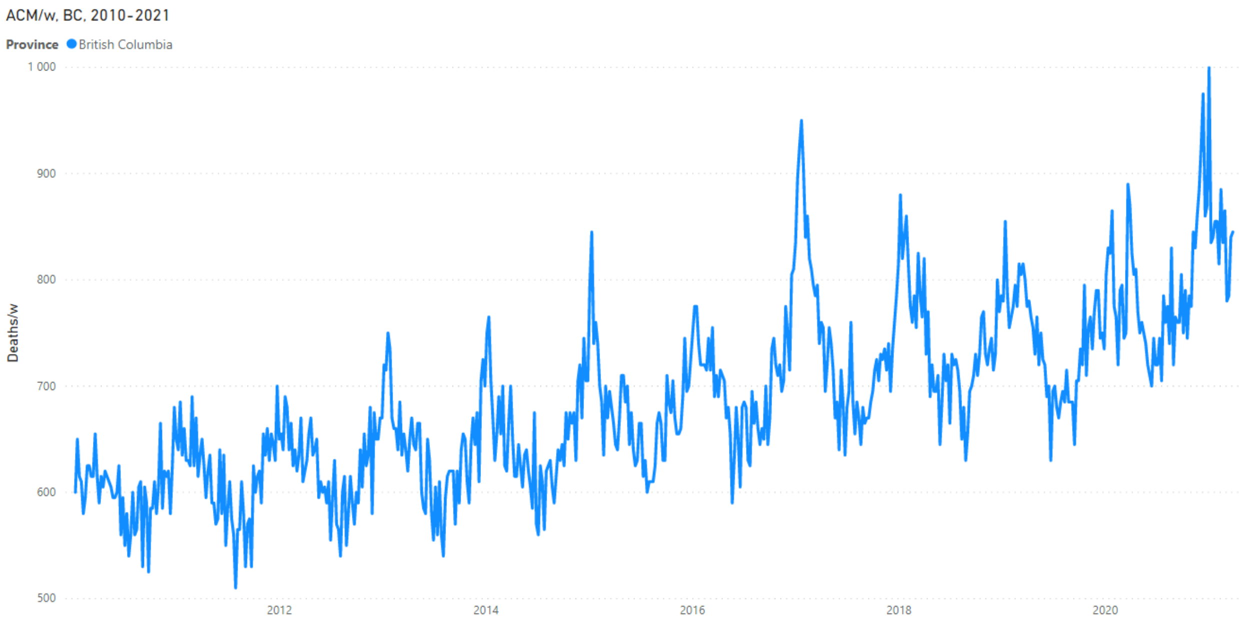


Figure 5-BC: All-cause mortality by week in British Columbia from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

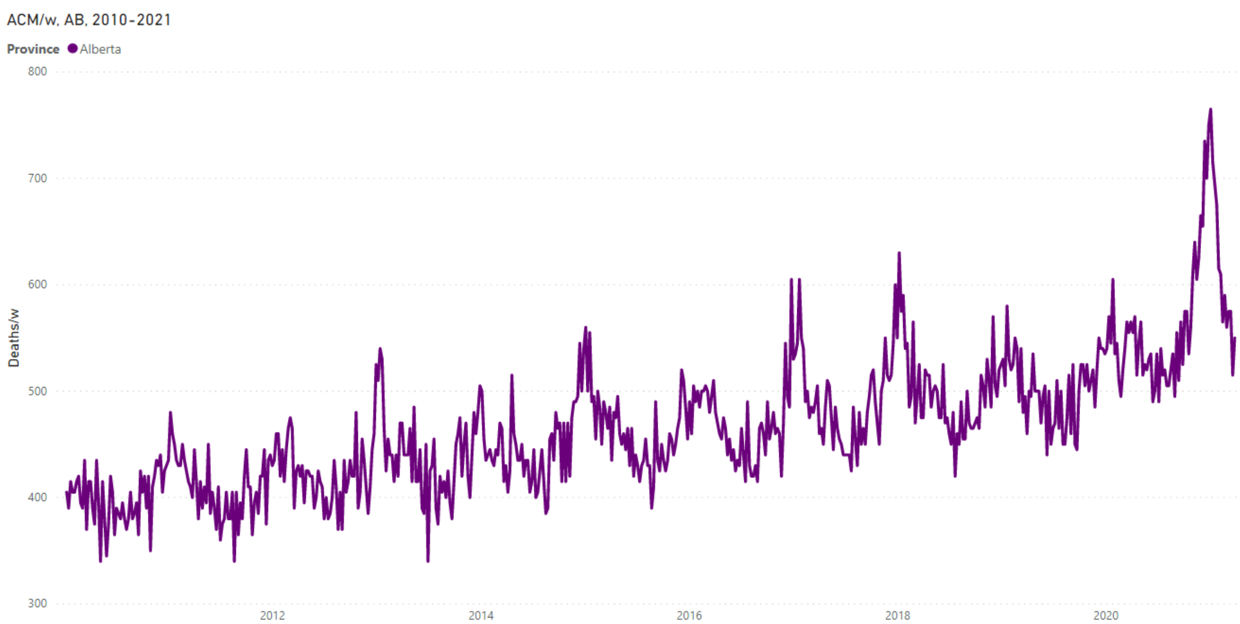


Figure 5-AB: All-cause mortality by week in Alberta from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

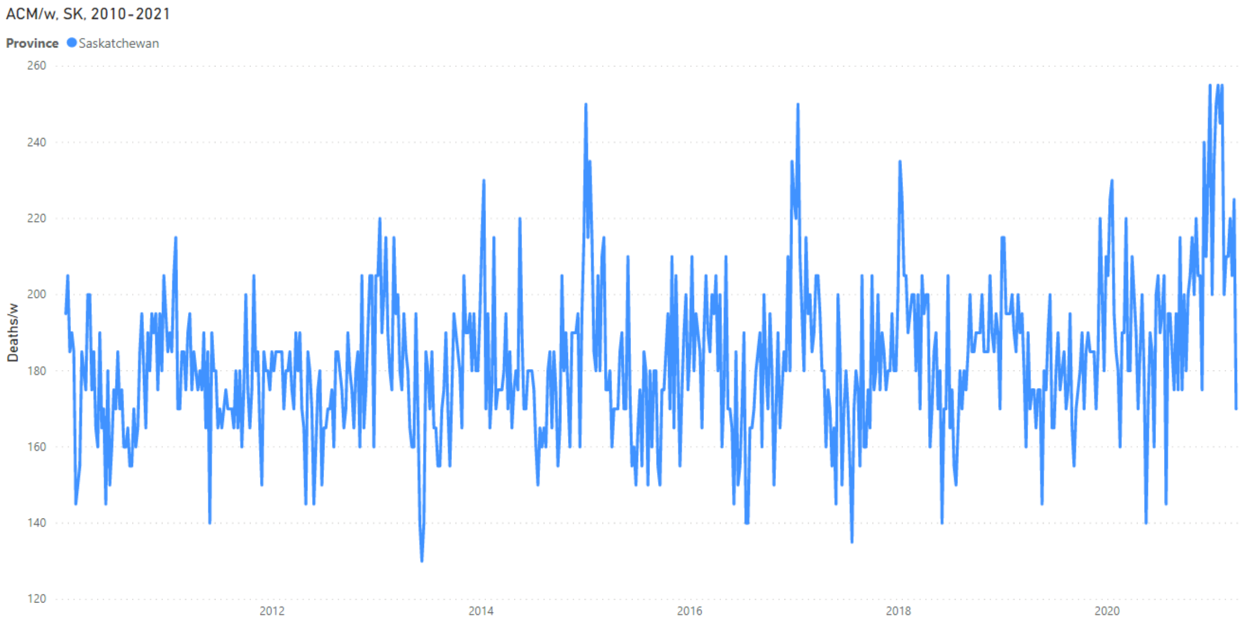


Figure 5-SK: All-cause mortality by week in Saskatchewan from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

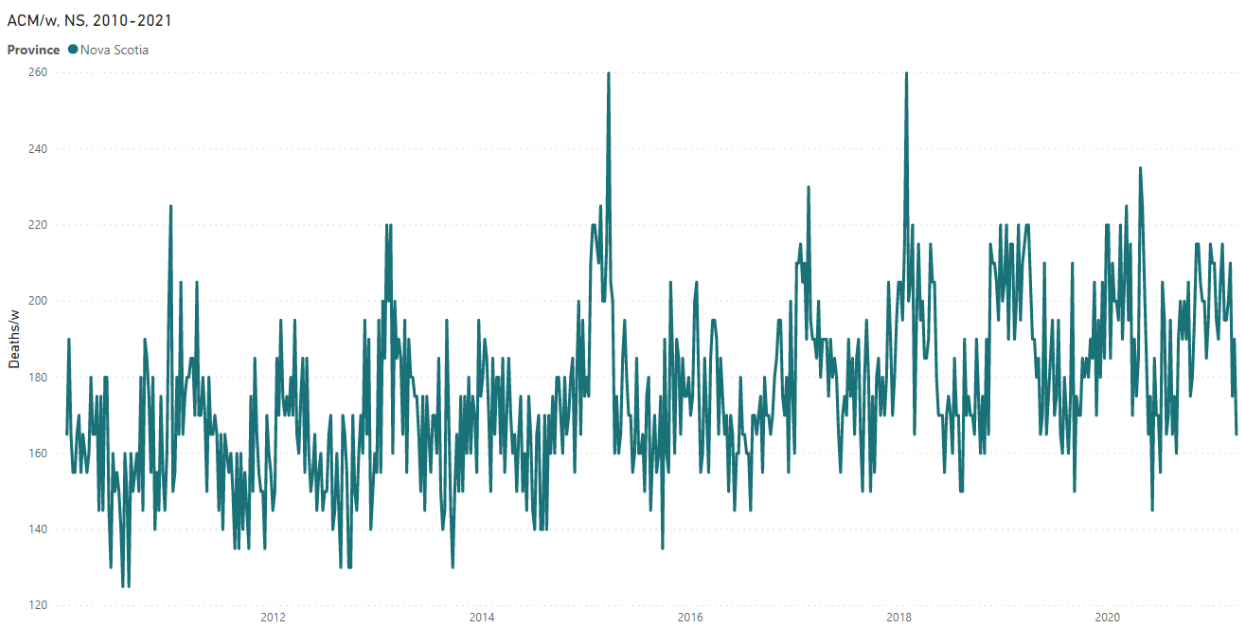


Figure 5-NS: All-cause mortality by week in Nova Scotia from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

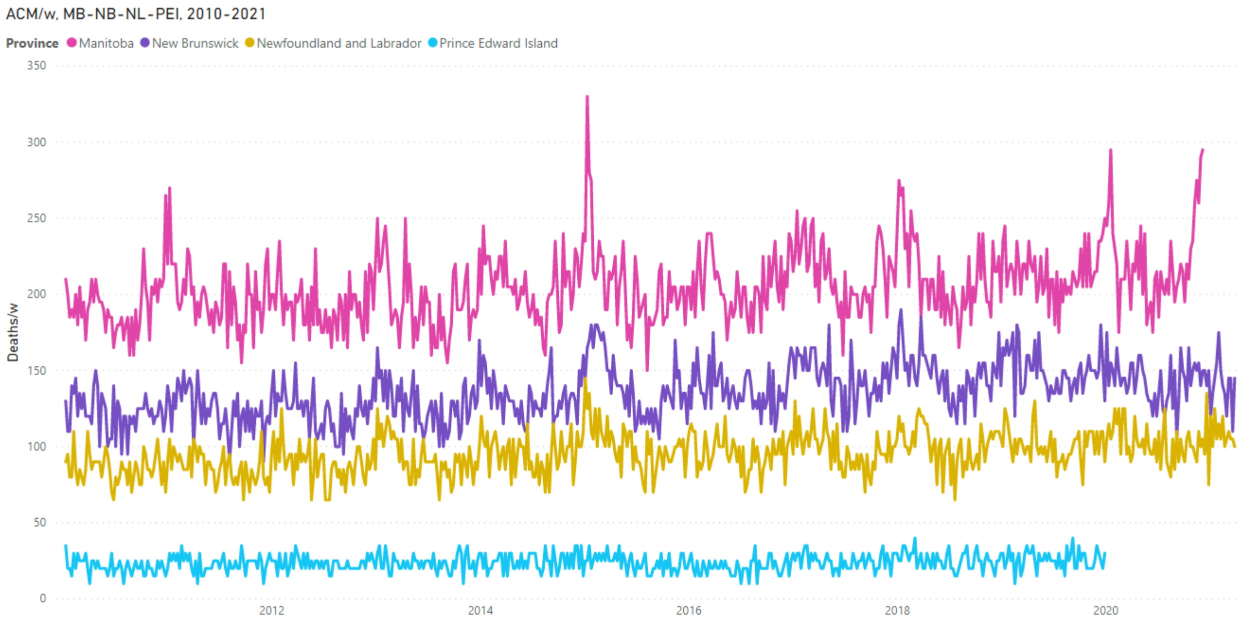


Figure 5-MB-NB-NL-PEI: All-cause mortality by week from 2010 to 2021 for, top to bottom, Manitoba (MB), New Brunswick (NB), Newfoundland and Labrador (NL) and Prince Edward Island (PEI). Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

Most notably:

- The "C"-feature ("covid-peak") for Quebec is exceptionally large among all provinces. Among other factors, Quebec care-home workers are known to have abandoned their locked-in patients *en masse*, presumably out of fear, even leading to criminal investigations.[2]
- The "C"-feature ("covid-peak") for Ontario is also unambiguously anomalous, as a large feature of this magnitude and shape this late in the winter-mortality

[2] "Montreal police, coroner investigating owner of seniors' residence where 31 died in less than 1 month" by Colin Harris · CBC News · Posted: Apr 12, 2020 12:56 PM ET | Last Updated: April 13, 2020 (accessed 6 August 2021). https://www.cbc.ca/news/canada/montreal/covid-19-private-seniors-home-dorval-chsld-herron-1.5530327

season. There was also large-scale care-home negligence in Ontario, documented in investigative media articles and a military report.[3]

- The "C"-feature ("covid-peak") is present for British Columbia, indicating some measures-induced and treatment-induced deaths in care-homes and hospitals, but to a lesser degree than in Ontario and Quebec.
- The "2"-feature ("2nd wave") is massive in Alberta, which is exceptional among all provinces. The peak is twice as high as any other winter peak for Alberta in the decade 2010-2020. Alberta also has an exceptionally high summer-2020 mortality, relative to its prior-decade trend of summer-trough mean magnitudes.
- Both Ontario and Saskatchewan also have high summer-2020 mortalities, relative to their respective prior-decade trends of summer-trough mean magnitudes, and unusually large "2"-features ("2nd waves"), but not to the degree observed for Alberta.
- Most East coast provinces (NS, NL, PEI, not NB) have small-amplitude seasonal cycles of ACM; and none for which there are data (NS, NL, NB) have ACM/w that exhibits any evidence of a COVID-19 pandemic or disruption, none whatsoever (data is missing for PEI).

[3] "Military report reveals what sector has long known: Ontario's nursing homes are in trouble" by Adam Carter · CBC News · Posted: May 27, 2020 4:00 AM ET | Last Updated: May 27, 2020 (accessed 6 August 2021). https://www.cbc.ca/news/canada/toronto/military-long-term-care-home-report-covid-ontario-1.5585844

3.4 Analysis of ACM/w by age group and by sex

The plots of ACM/w, from January 2010 through March 2021, for Canada, by age group (age at time of death), for the four age groups (0-44, 45-64, 65-84, 85+ years), are as follows.

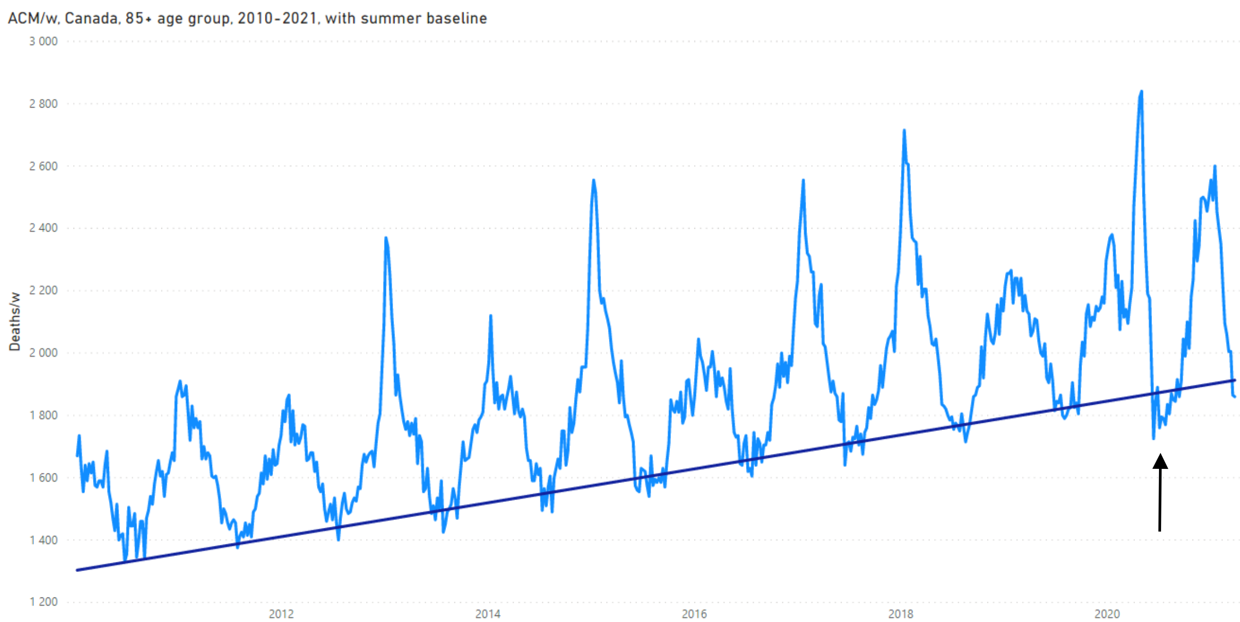


Figure 6a: All-cause mortality by week in Canada for the 85+ years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. The linear trend-line is a least-squares fit to the summer troughs for summer-2013 through summer-2019, using the following summer trough weeks: 2013-weeks 24-37, 2014-weeks 28-33, 2015-weeks 25-38, 2016-weeks 24-34, 2017-weeks 24-33, 2018-weeks 27-35, 2019-weeks 26-38. The arrow indicates a feature discussed in the text. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.



Figure 6b: All-cause mortality by week in Canada for the 65-84 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

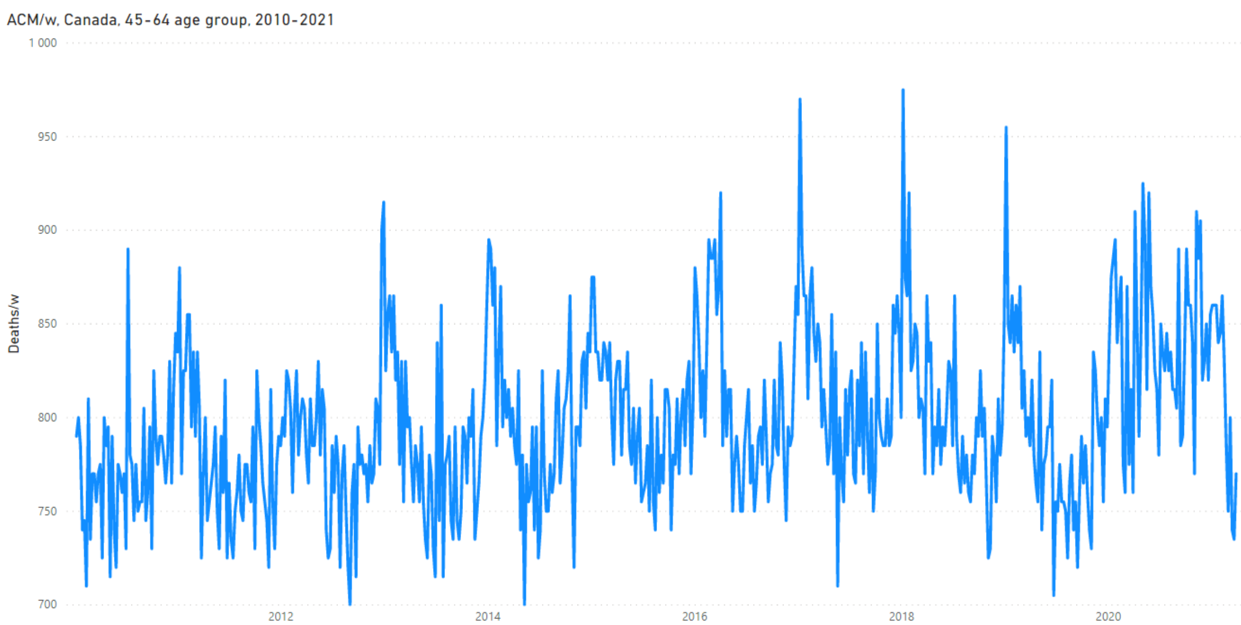


Figure 6c: All-cause mortality by week in Canada for the 45-64 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

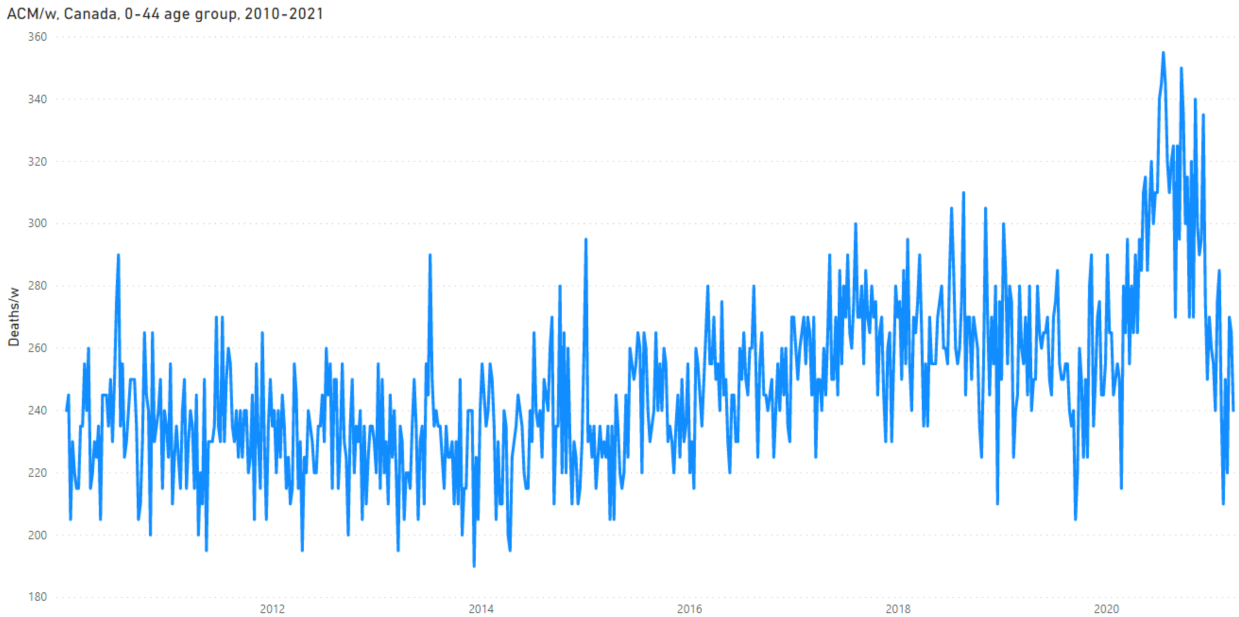


Figure 6d: All-cause mortality by week in Canada for the 0-44 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

Several observations can be made about the ACM/w data shown in Figures 6a through 6d, as follows:

- The amplitude (summer mean baseline value to winter maximum) of the seasonal variations in ACM/w, normalised by the summer mean baseline value, varies significantly with age, approximately as: near-zero for 0-44 years (no seasonal variation), 20% for 45-64 years, 30% for 65-84 years, and some 60% for 85+ years. The causes of increased winter deaths are more effective in the elderly, and all the more the older one gets.
- The patterns ("fingerprints") of ACM/w are essentially identical for the 85+ and 65-84 years age groups, prior to the COVID-period (prior to 11 March 2020). See plots of direct comparisons in the Appendix. This suggests that the causes for

increased winter deaths, and their timing, are the same in the two age groups, normally, where only the magnitude for the age group is affected by increased generalized frailty in the most elderly. Stated differently: One age group does not die of different causes than the other, regarding the increased likelihood of death in the winter.

- The latter point, regarding virtually identical intra-season time-structures, for each given season in the two age groups of the most elderly, including in the COVID-period, suggests that the driver of increased winter deaths is synchronized by the same cause(s) for the two age groups, which precludes vitamin deficiency, cancer, heart attacks and strokes, acting alone, but does not preclude weather, sudden societal or economic or institutional changes, sudden geological events, or sudden appearances of high-concentrations of pathogens in the living environments.
- The “C”-feature (“covid-peak”) in the ACM/w of the 85+ years age group (Figure 6a) is anomalous, relative to known ACM by time data of the last many decades for European and North American jurisdictions. Its dramatic drop occurs in a mere 6 weeks (as does its rise), during the weeks of 2 May 2020 to 13 June 2020, to summer-2020 values that are significantly below the linear trend-line for mean summer-trough values for summers 2013 through 2019 (Figure 6a).
- As such, the “S”-feature in the ACM/w of the 85+ years age group (Figure 6a) is equally anomalous. Why would 85+ year olds in Canada become relatively impervious to dying in the summer of 2020, in mid pandemic, between the presumed first and second waves of death? Our interpretation is: The deaths of

many 85+ year olds were artificially accelerated, at a time when seasonal VRD transmission is low, so that their deaths were not spread out into the following summer and fall, as would normally be the case.

- Another large anomaly, which should be considered a national public health catastrophe of historic proportion but is virtually absent from the media and government-official pronouncements, is shown in Figure 6d, for the 0-44 years age group. Here, we see a significant increase in deaths, from a pre-COVID-period plateau value of approximately 260 deaths/w to a summer-2020 value of approximately 320 death/w, lasting at least 28 weeks, into the start of December 2020. The peak corresponds to approximately 2,000 excess deaths in this 0-44 years age group in Canada, following the WHO pronouncement of a pandemic.
- The latter deaths cannot be ascribed to COVID-19 because the presumed disease virtually does not kill in this age group, and there is little transmission of VRDs in summer months. A similar but lesser relative increase in summer-2020 deaths occurs in the 45-64 years age group (Figure 6c).

The COVID-period excess deaths in the younger age groups can be further explored by sex, and by province. Relevant plots of ACM/w are as follows, for the 0-44 years age group, first for Canada, then select provinces.

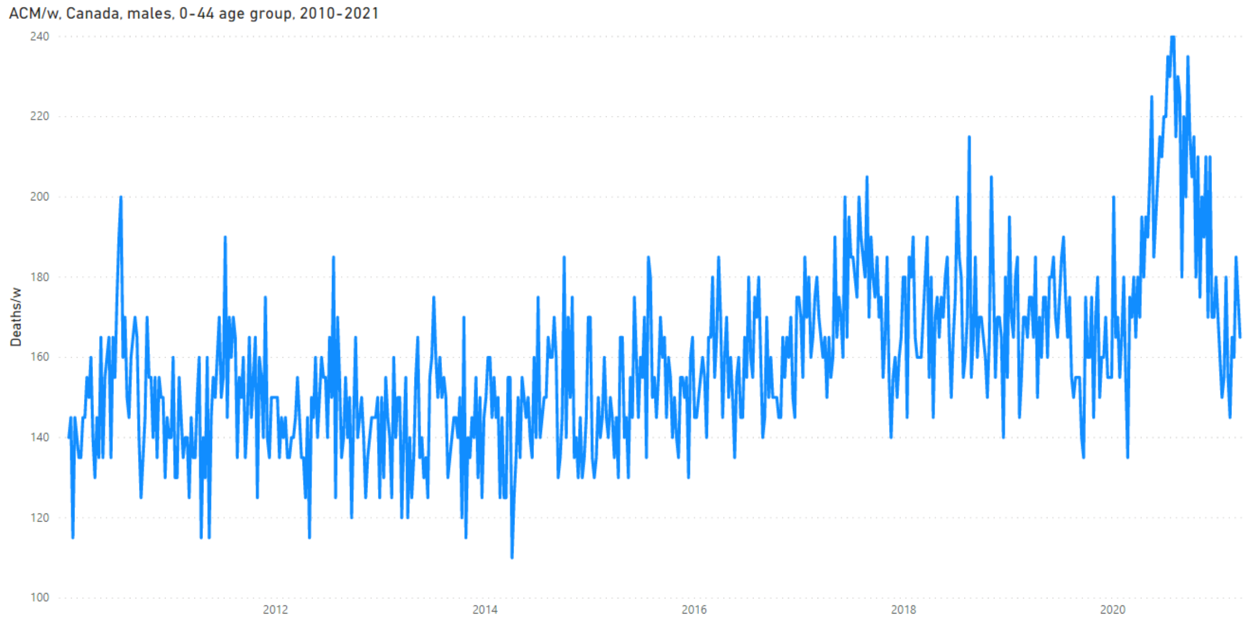


Figure 7a: All-cause mortality by week in Canada for males of the 0-44 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

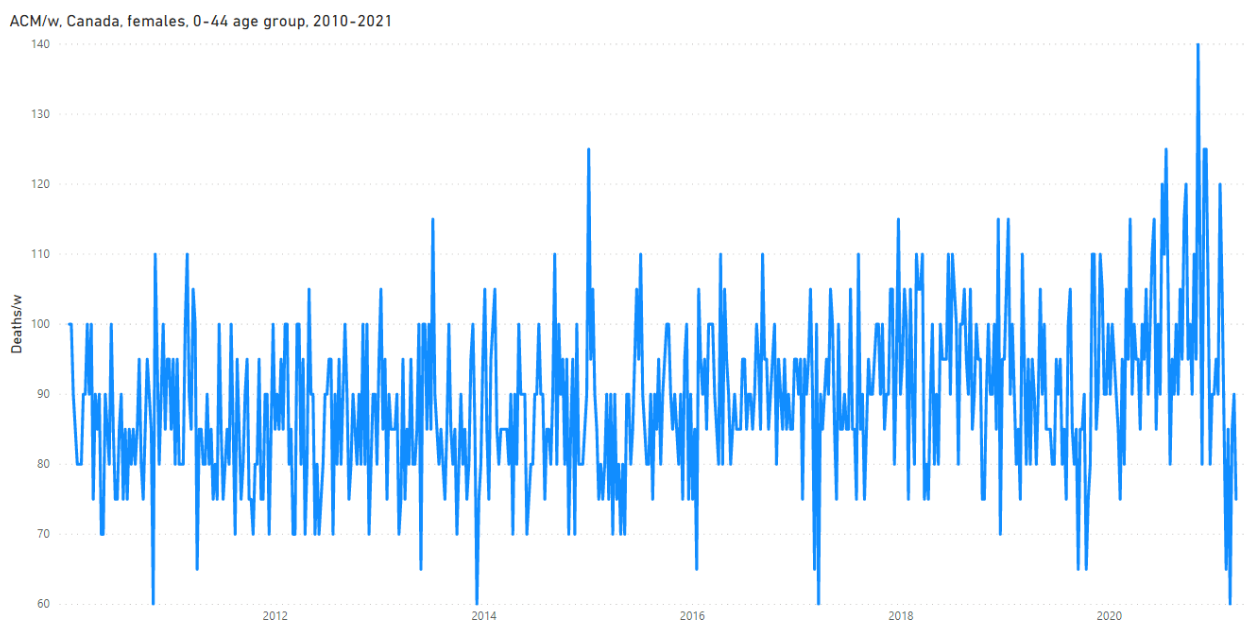


Figure 7b: All-cause mortality by week in Canada for females of the 0-44 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

Figures 7a & 7b show that, generally in the last decade, young Canadian males are almost twice (approximately 1.7 times) as likely to die of any cause compared to young Canadian females (0-44 years age group).

These figures (Figures 7a & 7b) also show that the excess summer-2020 deaths seen in this age group at the national level (Figure 6d) is almost entirely due to male deaths. This is also true for all the provinces that exhibit this feature in the 0-44 years age group. Virtually only males contribute to these excess deaths.

Next, we examine 0-44 years age group male deaths by province, as follows.

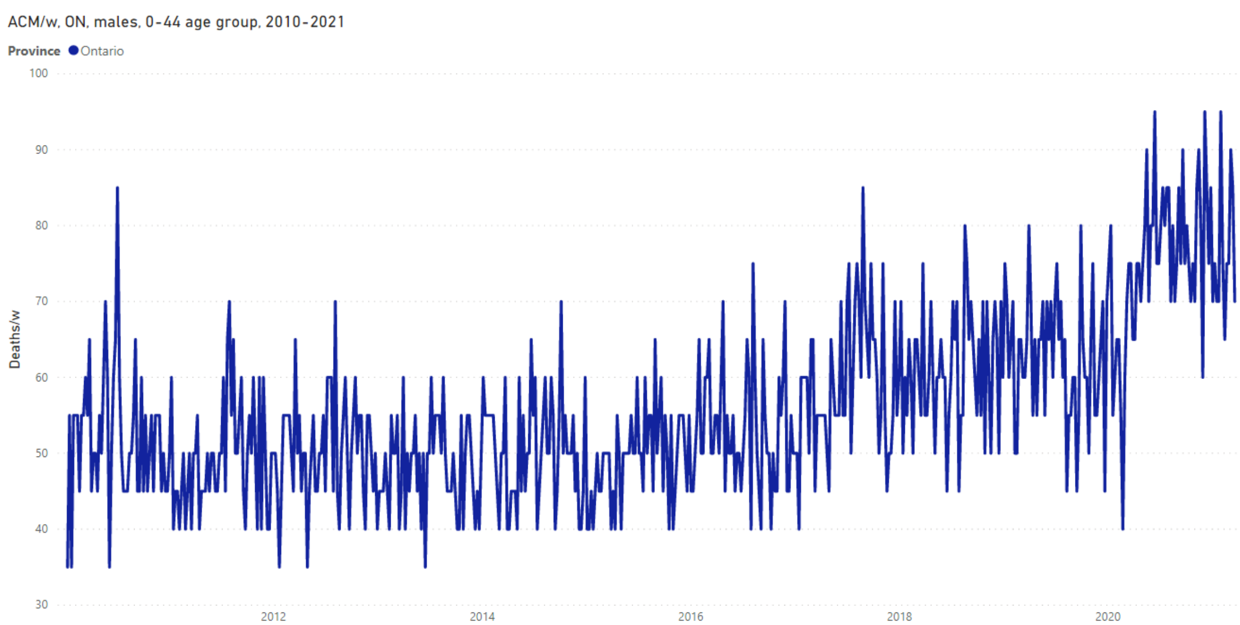


Figure 8-ON: All-cause mortality by week in Ontario for males of the 0-44 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

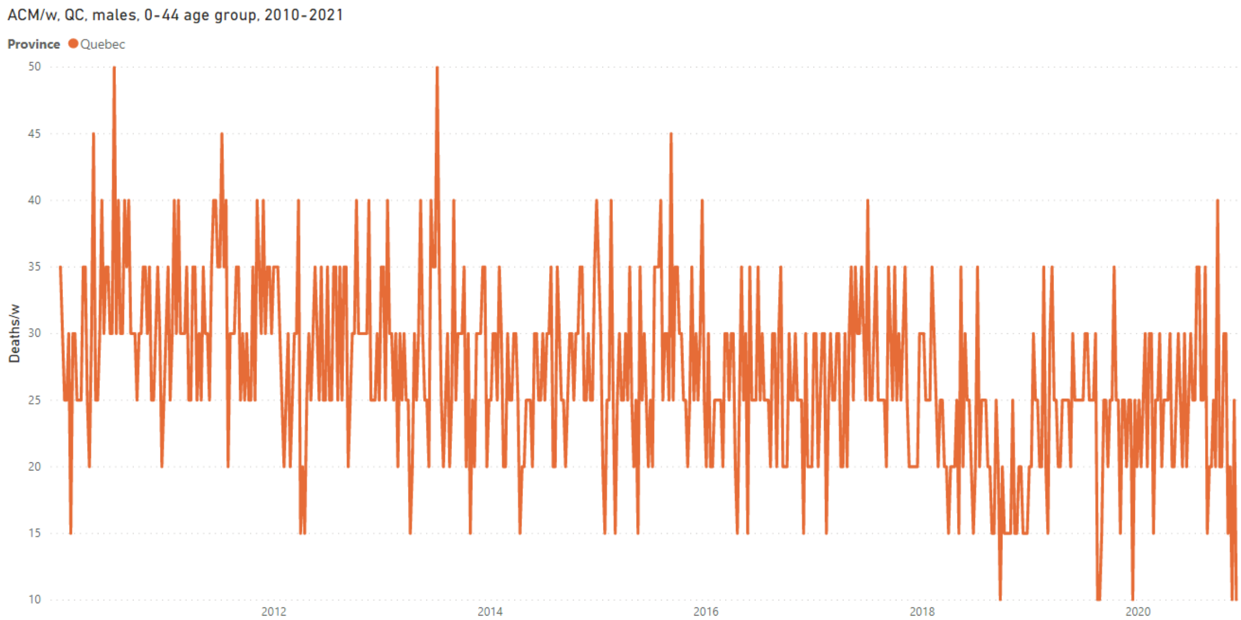


Figure 8-QC: All-cause mortality by week in Quebec for males of the 0-44 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

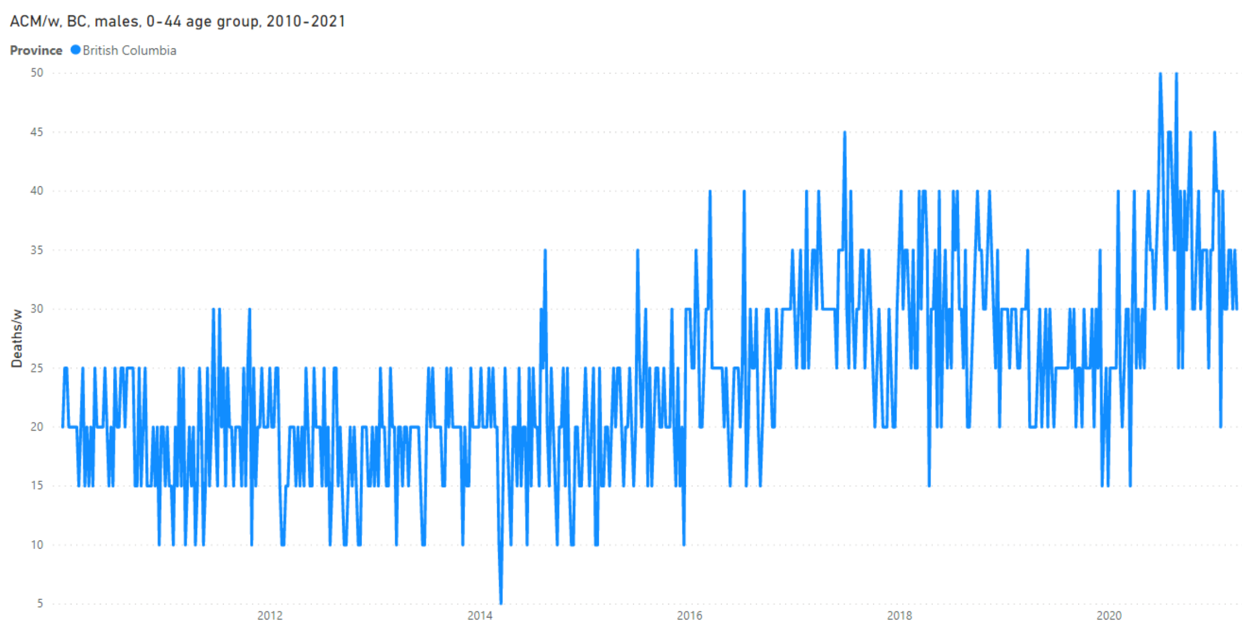


Figure 8-BC: All-cause mortality by week in British Columbia for males of the 0-44 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

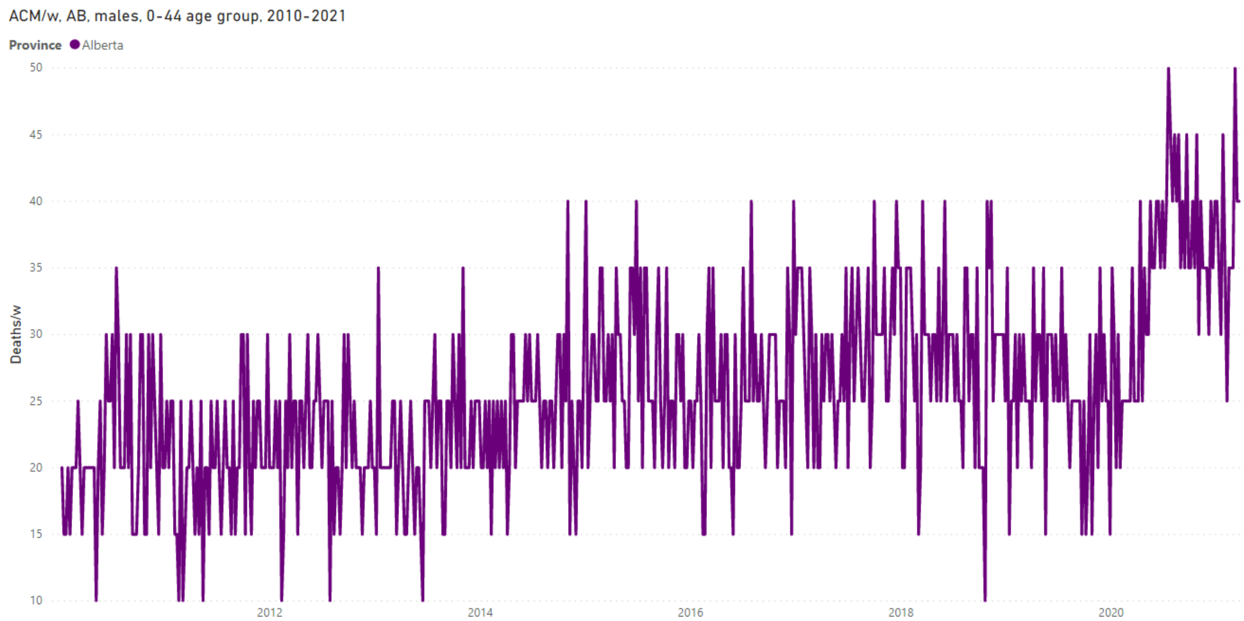


Figure 8-AB: All-cause mortality by week in Alberta for males of the 0-44 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

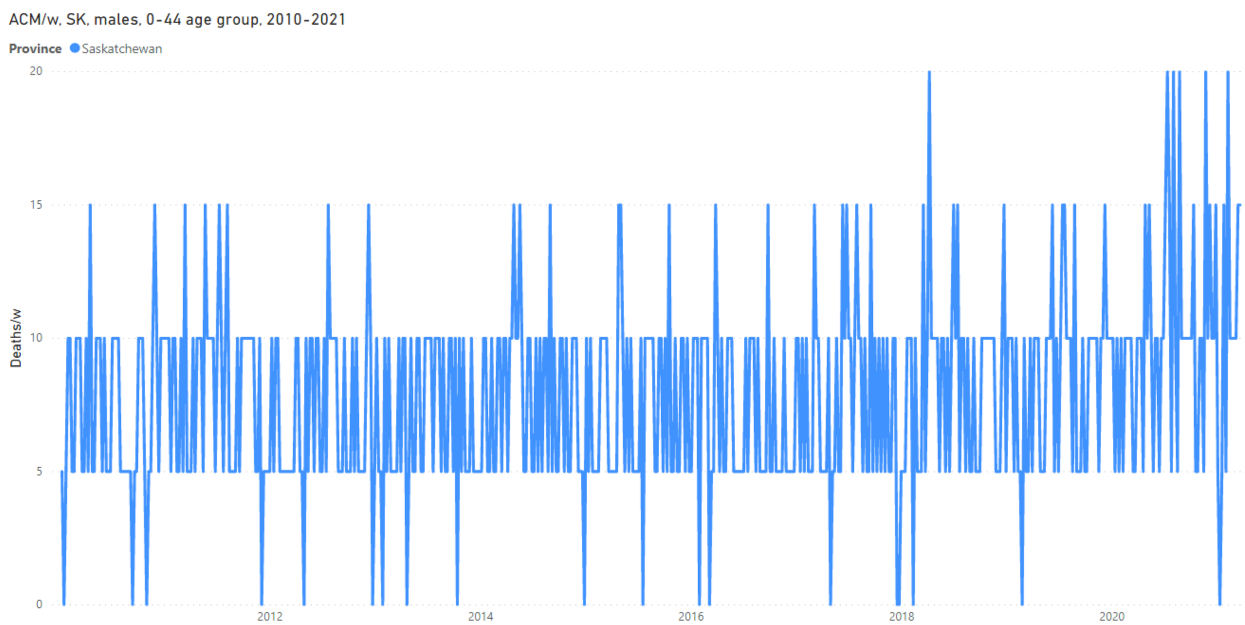


Figure 8-SK: All-cause mortality by week in Saskatchewan for males of the 0-44 years age group, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021), as described in section 2.

Thus we see that the phenomenon of excess 0-44 years age group male deaths is present in the large-population provinces, and in Saskatchewan and Manitoba (not shown), but exceptionally not present in Quebec.

Did the presumed SARS-CoV-2 virus decide not to act in this way in the province of Quebec, or is there another explanation? Our interpretation is that the excess deaths in males of the 0-44 years age group arise from the stress of the large-scale and continued societal and economic responses to the declared pandemic, and that the experienced stress in young men is lesser in Quebec because of significant cultural differences with Anglophone provinces, under conditions imposed by all provincial governments.

4. Discussion

4.1 Regarding pandemics

As noted above, the intra-seasonal and inter-seasonal time structures and the jurisdictional homogeneity in ACM by time, up to continental geographical scales for mid-latitudes, in unperturbed societies (unperturbed by sudden changes tied to world wars, or by sudden global “pandemic response” reorganizations), set constraints regarding the possible causes of the seasonal phenomenon having high winter death rates. Precluded causes are: vitamin deficiency, cancer, heart attacks and strokes,

acting alone. Not precluded causes include: weather, sudden societal or economic or institutional changes, sudden geological events, sudden appearances of high-concentrations of pathogens in the living environments, or combinations thereof.

We would argue for “sudden appearances of high-concentrations of pathogens in the living environments”. The stability-in-air of aerosol particles is known to be controlled by absolute humidity in mid-latitudes (e.g., see Rancourt, 2020b, and references therein). We imagine summer background population mixing, and faster dry-season population mixing, of continually arising mutations of pathogens that transmit by suspended aerosols (i.e., the entire ecology of VRD viruses), followed by sudden low-absolute-humidity-induced winter-time increases of concentrations (in the built environment - individual homes to public spaces) of aerosols bearing all such pathogens.

The infections from the multitude of co-acting VRD viruses would be accompanied by an array of opportunistic bacterial co-infections, aided by the dry-air stress on respiratory tract tissues.

We believe that the genome-centered view of single unique viral mutations/variants explaining seasonal structures in ACM by time is too narrow and over-emphasized. The contributions from weather and from the large array of co-acting pathogens must be more relevant than the “particular-special-new-mutation/variant virologist’s view”, otherwise pandemics would be observed in ACM by time data, and they are not.

Simply put, the pandemic paradigm is a beautiful theory, which is greatly pleasing to the genome jockeys, but it is not supported by hard epidemiological data, and it has a great potential to cloud public health thinking by directing focus on a presumed pathogen-specific disease rather than identifying and addressing all the important aspects of a health crisis or chronic-disease circumstances.

In Canada at least, in the present article we have shown that no additional yearly or seasonal integrated mortality occurs in the COVID-period (Figures 1 & 2). There was no COVID-19 pandemic in Canada, which can be detected in ACM by time. It would be a fantasy to believe that Canada avoided the COVID-19 pandemic deaths by its hurried, differing and unproven pandemic response, such as to exactly bring the resulting net yearly and seasonal mortalities in line with the trend of the last decade (Figure 2).

4.2 Regarding the “C”-feature (“covid-peak”) in ACM by time

The occurrence of dramatic jurisdiction-to-jurisdiction differences (jurisdictional heterogeneity) in the magnitude (relative to summer baseline) of the “C”-feature (“covid-peak”) in ACM/w by province in Canada is diametrically opposite to all pre-COVID-period ACM by time data that we have examined for many jurisdictions (countries, regions, provinces, counties) in North America and Europe, over the many decades of available data.

Whereas pre-COVID-period integrated winter-burden mortalities (above linear summer baseline trends), normalized by mean summer baseline mortality or by jurisdictional population, are always relatively constant between jurisdictions, the “covid-peak” feature varies widely between jurisdictions in a given country, or between countries, often being undetectable or borderline detectable, versus extreme “hot spot” jurisdictions.

For France, we calculate that, on the basis of region-level jurisdictional divisions, the standard deviation of the “covid-peak” integrated magnitude normalized by population divided by the mean (s.d./mean) is 3-fold greater than the standard deviation for integrated winter-burden magnitude (integrated above the linear trend of summer-trough minimums) normalized by population divided by the mean (s.d./mean) (article in preparation).

We argue that such jurisdictional heterogeneity cannot be due to a VRD epidemic in an unperturbed society, because such a phenomenon has never previously occurred in the many decades since reliable data is available for many jurisdictions. Only an unusually large perturbation of the society can produce such a phenomenon.

We believe that it is not a coincidence that all the “covid-peaks” — in jurisdictions where they occur on both continents — started their sharp and sudden surges immediately (within 1 week or so) after the WHO’s 11 March 2020 pronouncement of a pandemic. We believe that viruses did not suddenly everywhere act on cue in response to the

WHO memo, in those jurisdictions where the “covid-peak” feature occurs in ACM by time.

4.3 Regarding the summer-2020 level and the “2”-feature (“2nd wave”) in ACM by time

By-province heterogeneity is also present in the summer-2020 level and in the “2”-feature (“2nd wave”) in the COVID-period of ACM/w in Canada (esp. for Alberta, Figure 5-AB).

It is unlikely that a same pandemic-causing virus acted alone to produce significant excess deaths in the summer-2020 period, relative to the linear trend of summer baseline values, irrespective of the magnitude of the preceding “covid-peak”: Ontario (Figure 5-ON), British Columbia (Figure 5-BC) and Alberta (Figure 5-AB), but not noticeably in Quebec (Figure 5-QC), for instance.

It is possible that the excess deaths in the summer-2020 period were induced by the societal disruption of the pandemic response (more below), without being associated with any VRD, except secondarily *via* the so-called “dry tinder” effect following a large “covid-peak”.

More strikingly, the “2”-feature (“2nd wave”) peak for Alberta is massive, compared to any other province, whereas no noticeable “covid-peak” occurs in this province (Figure 5-AB). A pandemic-causing virus cannot decide not to produce a “1st wave” but only a “2nd wave” in one province of a continuously connected country having similar provincial populations. Nothing like this has ever been observed, to our knowledge.

We argue that the “2”-feature (“2nd wave”) peak, occurring during a winter-season of expected increased mortality, has varying province-wise magnitudes because of the province to province differences in pandemic response, and province to province differences in population resilience against the stress of the imposed measures.

In short, like with the “covid-peak”, such jurisdictional heterogeneity cannot be the result of the genome of a particular viral pathogen. Such epidemiological heterogeneity of presumed VRD mortality has not previously been observed in North America or Europe in many decades of reliable ACM by time data. VRD viruses of any mutation or variety do not recognize jurisdictional boundaries and do not act so widely differently on similar populations on continuous territories. The large features of the ACM by time data for the COVID-period can only be explained by appealing to additional causal factors beyond the limited purview of virology.

4.4 Regarding age group specifics in ACM by time

The ACM/w in Canada for the 85+ years age group (Figure 6a) allowed us to partly unravel the complex and unusual behaviour of mortality in the COVID-period. As mentioned above, the sharp drop in its "covid-peak" connects to a summer-2020 having anomalously small mortality for this age group (Figure 6a).

This is all the more surprising in that the summer-2020 mortality for all age groups is anomalously large (Figure 1). Cumulatively, all ages have an anomalously large summer-2020 mortality, whereas the 85+ years age group has an anomalously small summer-2020 mortality. Mortality of younger Canadians increased, in a season that does not normally carry many VRD infections, whereas less mortality occurred for the most aged Canadians.

In the ACM/w data for the 85+ years age group (Figure 6a), the "covid-peak" followed by an anomalously small summer-2020 mortality, may be a most compelling example of the so-called "dry tinder" effect, in which successive winter-season mortalities are argued to be anti-correlated because a harsh winter leaves fewer frail elderly to die in the following winter. Whereas this postulated winter-to-winter anti-correlation is not easily discerned, except in earlier times when mortalities were larger (see mid-1940s to mid-1950s for France, Figure 1 of Rancourt, Baudin, Mercier, 2020), here (Figure 6a) we demonstrate the effect, within an exceptional year, in current times.

Finally, there is the anomalous mass mortality of young males in Canada, especially in Alberta but not in Quebec, in summer-2020 and into the fall (Figure 7, all parts). This ignored and silent epidemic is most likely not due to any VRD, and merits an independent investigation in its own right.

4.5 Regarding causes of response-induced deaths

We seek to describe plausible mechanisms whereby sudden disruptions in society can induce deaths, or reduce deaths at later times, without necessarily significantly changing the yearly or seasonal death burden compared to a decadal trend, following (Rancourt, 2020) (Rancourt, Baudin, Mercier, 2020).

We propose that there are three large categories of such plausible mechanisms:

- Medical response, treatment and palliative protocols, adopted at the onset of the proclaimed and media-hyped pandemic.
- Pandemic response, public health measures, institutional protocols (esp. schools, care homes, and hospitals), economic upheaval, lockdowns, curfews, self-quarantine, etc.
- Policies of denial of medical treatment, such as refusal to admit elderly persons into hospital care, or transfers of patients out of hospital care.

In France, for example, as in many other countries, starting in March 2020 there were tremendous social and medical disruptions, not planned or previously applied. The national lockdown in-effect was a "stay-at-home" order, including not visiting the family physician, and to call the emergency services only in cases of breathing difficulty, which was by itself a dangerous recommendation as people presenting those symptoms were usually already in a late stage of disease, often admitted to hospital directly into the intensive care unit. This reckless protocol directed by health authorities concerned not only COVID-19, but generally all medical conditions since people were asked to stay at home, to not visit their general practitioners, nor to show up at hospitals (to avoid an unmanageable institutional burden). Another statement from the health authorities was that no treatment exists for COVID-19: people were told to take *Doliprane*® (acetaminophen) in case of symptoms; and healthcare professionals were denied using or attempting any medical protocol. This caused abandonment of medical care by the general population and by healthcare professionals, following the official recommendations. The official recommendations thereby may have promoted excessive and dangerous self-medication with over-the-counter substances such as *Doliprane*® and analogous drugs. Signatures of the unprecedented perturbation in the healthcare system include changes in specific drug usage and consumption in 2020, such as significant drops in the use of antibiotics and significant increases in the use of psychoactive drugs (Chaillot, 2020) (and our article in preparation). One specific example is the *Rivotril*® drug (clonazepam) in its injectable form, which by decree[4] could exceptionally by used from 23 March to 15 April 2020 without marketing authorization to

[4] Décret N° 2020-293 Du 23 Mars 2020 Prescrivant Les Mesures Générales Nécessaires Pour Faire Face à l'épidémie de Covid-19 Dans Le Cadre de l'état d'urgence Sanitaire.; 2020. https://www.legifrance.gouv.fr/loda/id/LEGIARTI000041767762/2020-03-29/

terminate patients affected or likely to be affected by SARS-CoV-2 if their health status justified it, and which showed an increase of more than 200% in April 2020 compared to the mean over January 2017 to February 2020 (Chaillot, 2020).

In the USA, the early over-use of mechanical ventilators is a well-studied aspect of deadly COVID-19 medical responses (Richardson et al., 2020).

In addition, and in Canada, the unprecedented strict mass quarantine and isolation of both sick and healthy elderly people, together and separately, would have caused the deaths of many of them, and is probably a main cause of the “covid-peak” event in Canada, where a great majority of COVID-19-assigned deaths occurred in care homes for the elderly (Clarke, 2021):

> During the first wave of the pandemic (March through August 2020), residents of nursing and seniors’ homes accounted for more than 80% of all reported COVID-19 deaths (ref). […] By mid-December (partway through the second wave that lasted from September 2020 through February 2021), there were about 44,000 cases and 9,200 deaths in nursing and seniors’ homes (ref). As of early March 2021, reports indicated that nursing and seniors’ homes continued to account for the greatest proportion of outbreak-related cases and deaths, representing about 7% of all cases and more than 50% of all deaths (refs).

By the said mass quarantine in care homes and establishments, Canadian provincial institutions isolated vulnerable elderly persons from their families, limited movements within establishments, often confining individuals to their rooms or beds for days and

weeks if not months, reduced the staff and allowed staff to be absent, forced staff to adopt extreme measures such as masks, shields and gloves, which can induce a measure of fear or terror, created a general atmosphere of danger, and prevented air circulation by locking doors and windows, and by preventing ingoing and outgoing traffic except for essential services (Campbell, 2020; Comas-Herrera, Fernandez, *et al.*, 2020; Wu, 2020).

This would have both: retained the pathogen-bearing aerosol particles suspended in the air without their evacuation (Morawska and Milton, 2020); and induced psychological stress in the residents.

Psychological stress is known:

i. to be a major factor causing diseases, including immune response dysfunction, depression, cardiovascular disease and cancer (Cohen, Janicki-Deverts and Miller, 2007),
ii. to be a dominant factor in making an individual susceptible to viral respiratory diseases, in terms of intensity of the infection (Cohen, Tyrrell and Smith, 1991), and
iii. to have more deleterious effects in elderly persons than in younger persons (Prenderville *et al.*, 2015).

Furthermore, social isolation itself, in addition to individual psychological stress, is known to have an added impact on the said susceptibility to viral respiratory disease (Cohen *et al.*, 1997).

Furthermore, there is a longer term “abandonment of life” phenomenon that occurs with imposed extended isolations of elderly persons, the so-called “*glissement*” syndrome (or “slipping away syndrome” or “geriatric failure to thrive”), which is analogous to depression (Robertson and Montagnini, 2004; Clegg *et al.*, 2013; Steptoe *et al.*, 2013; Ong, Uchino and Wethington, 2016).

The suddenly applied national policy of forced quarantine and the psychological stress it generated on fragile elderly people would have been a contributor in the decrease of efficiency of immune system response to a viral respiratory disease (Comas-Herrera, Zalakaín, *et al.*, 2020) and this is a probable explanation for much of the mortality in the “covid-peak” and in the “2nd wave”. The same mechanism would operate in any setting (facility, group home, home, hospital) where persons with health vulnerabilities are isolated and susceptible to psychological stress.

Whereas care homes are institutional environments that are extremely susceptible to epidemics, whereas VRD epidemics in care homes are common and this is well known (Utsumi et al., 2010), and whereas the best recommendation to prevent the spread of a VRD epidemic in a care home is vigilant and early diagnosis of cases of clinically ill infected individuals followed by rapid effective treatment and isolation/distancing of

those individuals (Loeb et al., 2000) (Bowles et al., 2003), therefore it is important to note that the opposite was done in Canadian care homes: no surveillance for emergent clinical infections, no treatment or search for treatment, no targeted removal/distancing or isolation of the clinically ill infected individuals, and universal lockdown of all residents. Even antibiotic treatment of bacterial co-infections may have been in-effect denied, as appears to have been the case in France (as mentioned above).

Rancourt recently summarized the situation this way:[5]

> The mechanism that made care homes and institutions for sick and elderly persons into killing fields includes the following elements (refs):
>
> - infection seeding by hospital transfers into the care homes
> - universal lockdowns of the care homes
> - denied specialized medical treatment to the residents of the care homes
> - reduced staffing and staff abandonment in the care homes, and negligence
> - collateral effects of the universal lockdown of the care homes: extreme social isolation, psychological stress, reduced aerosol-exhaust ventilation, lost oversight of the institutions by family-members

We can add the use of *Rivotril*® (in France), which would have terminated some elderly patients with breathing difficulties, and other changes in treatment practices (see above).

[5] "The Great VIRAL Debate: Dr Rancourt's Closing Statement" by Denis Rancourt, *Off-Guardian* (10 November 2020) (Accessed on 6 August 2021). https://off-guardian.org/2020/11/10/the-great-viral-debate-dr-rancourts-closing-statement/

4.6 Would there have been fewer deaths?

Although we have shown that there was no pandemic, nonetheless, there are year to year variations in mortality in non-pandemic years, and a valid question remains: Would fewer immediate and later deaths have resulted in the absence of the pandemic response?

We conclude that the answer is “yes”. The “covid-peak” was palpably induced by the pandemic response, at a time in the long-term seasonal cycle when there is always a decline in ACM by time. It was followed by an anomalously small mortality for the 85+ years age group, showing that deaths were accelerated in this age group. Likewise, the mortality of young males (0-44 years) has a large increase in the summer-2020, and into the fall, a phenomenon never before seen, which cannot be due to a VRD pathogen.

5. Concluding comments: Missing self-evaluation

We proved that there was no pandemic in the COVID-period in Canada, if the concept of a pandemic means anything. We showed strong evidence that the pandemic response was so aggressive and ill-advised as to have large negative health consequences, identified in ACM by time.

Although there was no pandemic, our analysis of the ACM by time data suggests that the pandemic response in Canada was a reckless and deadly fiasco. Had there been a particularly virulent pathogen, this level of government and institutional negligence, based on the international trend in attitudes and on political motives, would not have been possible.

There is no concrete evidence that the provincial and federal governments have learned any lesson from what was a massive public health blunder. On the contrary, there is every sign that governments continue to have a siloed approach based entirely on vaccine programs and ineffective personal hygiene regulations, while ignoring the science relevant to what actually occurred in Canadian care homes, and while avoiding strategies to start to address what actually occurred, and is occurring.

A first and immediate step should be to trash the pandemic-response methods that were implemented after the WHO's declaration of a pandemic, and to develop expertise-based national skepticism about such declarations and their accompanying recommendations.

We hope that our analysis will be useful to public health policy reviewers, and that the needed serious in-depth critical review of the government and medical responses will be undertaken, one way or another. We further hope that this will be done with transparency and accountability, and that it will include broad consultations.

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Appendix: ACM/w normalized by population, and comparisons

In this appendix, we show various plots of ACM/w, normalized by population, and various plots comparing ACM/w data, by province, and by age group.

Statistics Canada (StatCan) is the national statistical office of the country. The all-cause mortality (ACM) and the population (pop) data used in this appendix were retrieved from the StatCan database. The following table shows the characteristics of the data:

Data	Geography	Period	Frequency	Source
ACM	Canada Province Territory	2010-2021*	Weekly	StatCan, 2021
Population	Canada Province Territory	1971-2020	Annual	StatCan, 2020

* At the date of access, data were available from week-1 of 2010 (beginning of January) to week-17 of 2021 (end of April). In the following figures, we show the data until week-12 of 2021 (end of March), because the data are not consolidated in later weeks, which gives a large artifact (anomalous drop in mortality).

Moreover, data can be retrieved by sex (males/females) or by age group. For the population data, the age groups are year by year from 0 to 99 years-old, and the last group is 100 years-old and over. For the ACM data, the age groups are as follows:

- 0-44 years-old
- 45-64 years-old
- 65-84 years-old
- 85 years-old and over

The population is estimated on July 1st of each year. The ACM/w of one calendar year has been normalized by the population of that calendar year (ACM/pop/w). The only exception is the year 2021, as there are no population estimates for that year, the ACM/w has been normalized by the population estimates for 2020.

Sources

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2020--StatCan : Statistics Canada (2020). Table 17-10-0005-01 Population estimates on July 1st, by age and sex https://doi.org/10.25318/1710000501-eng (accessed 31 July 2021)

Appendix Figures

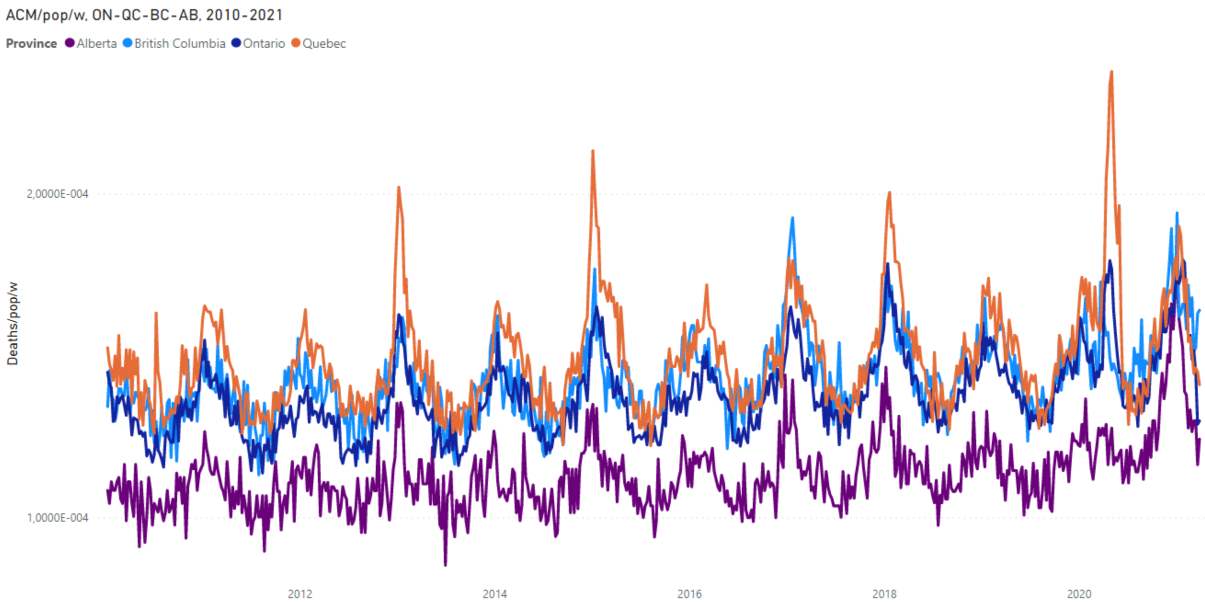


Figure A1: All-cause mortality by population by week in Ontario, Quebec, British Columbia and Alberta from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

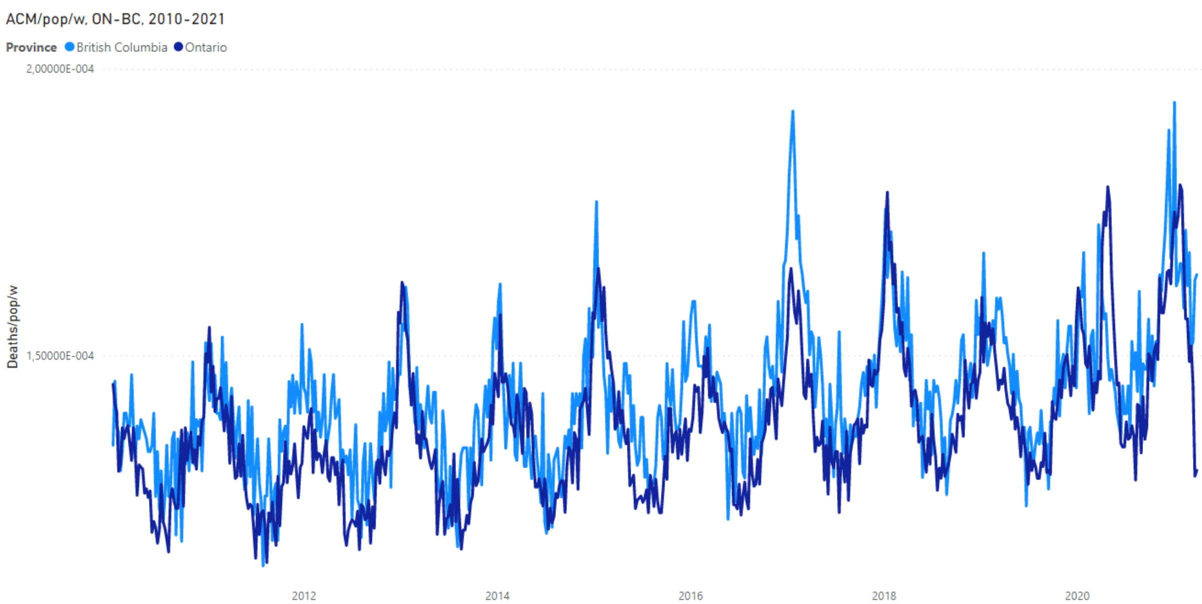


Figure A2: All-cause mortality by population by week in Ontario and British Columbia from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

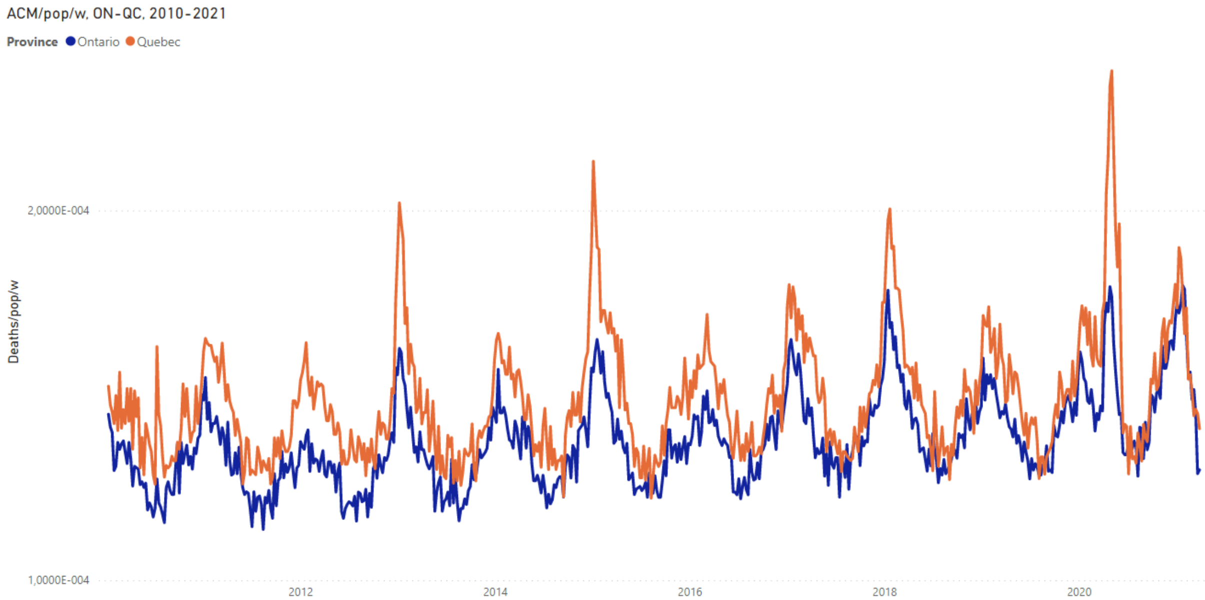


Figure A3: All-cause mortality by population by week in Ontario and Quebec from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

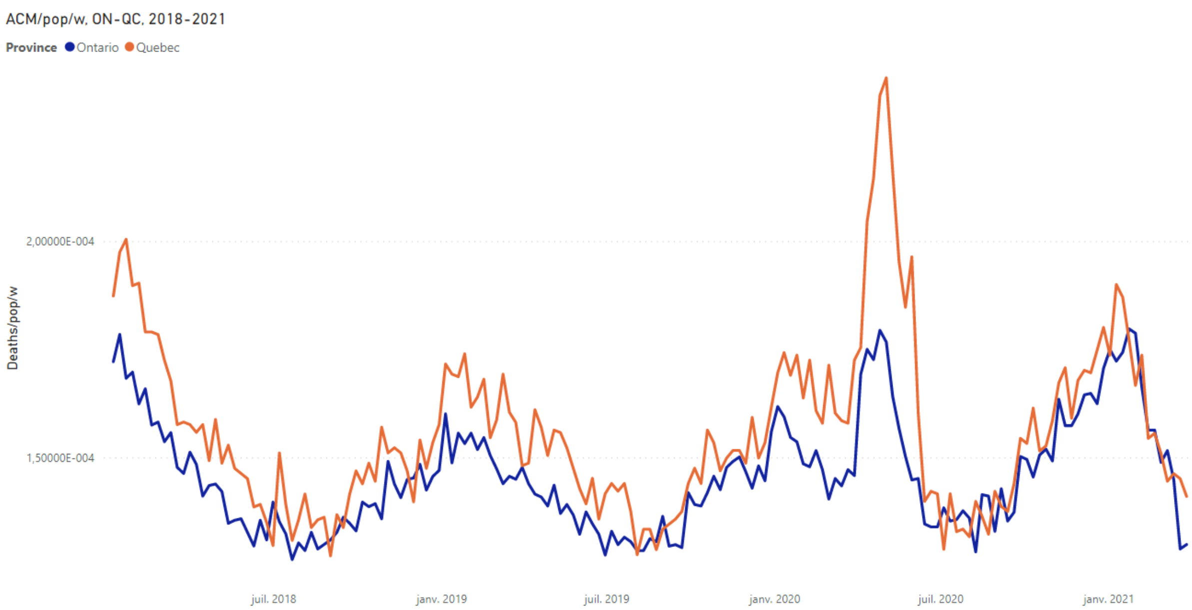


Figure A4: All-cause mortality by population by week in Ontario and Quebec from 2018 to 2021. Data are displayed from January 2018 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

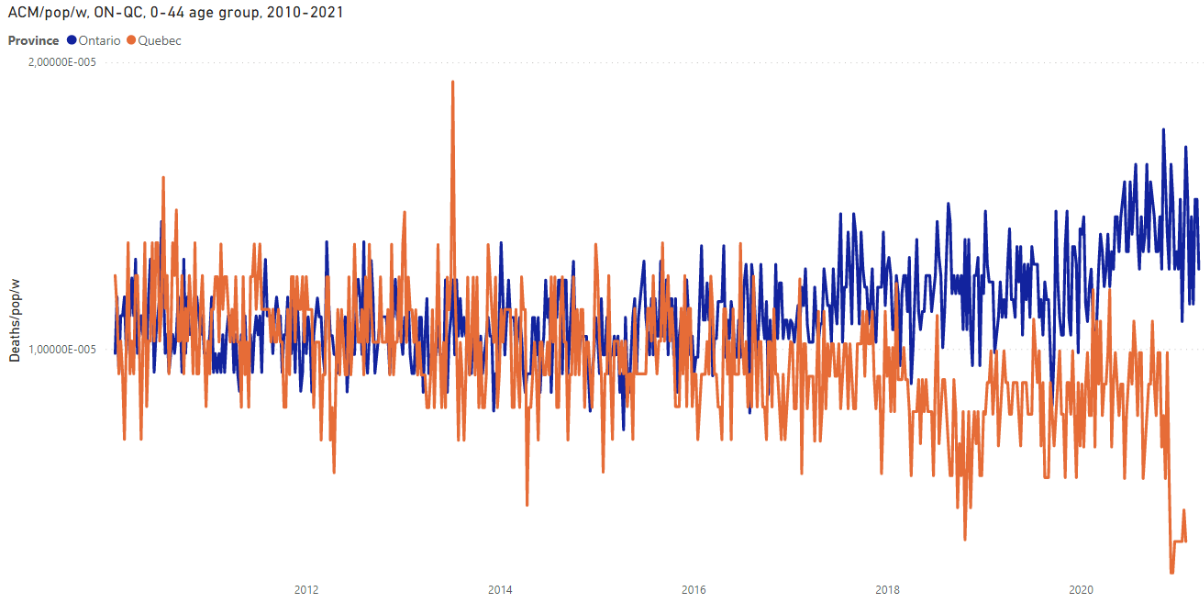


Figure A5: All-cause mortality by population by week in Ontario and Quebec for the 0-44 age group, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

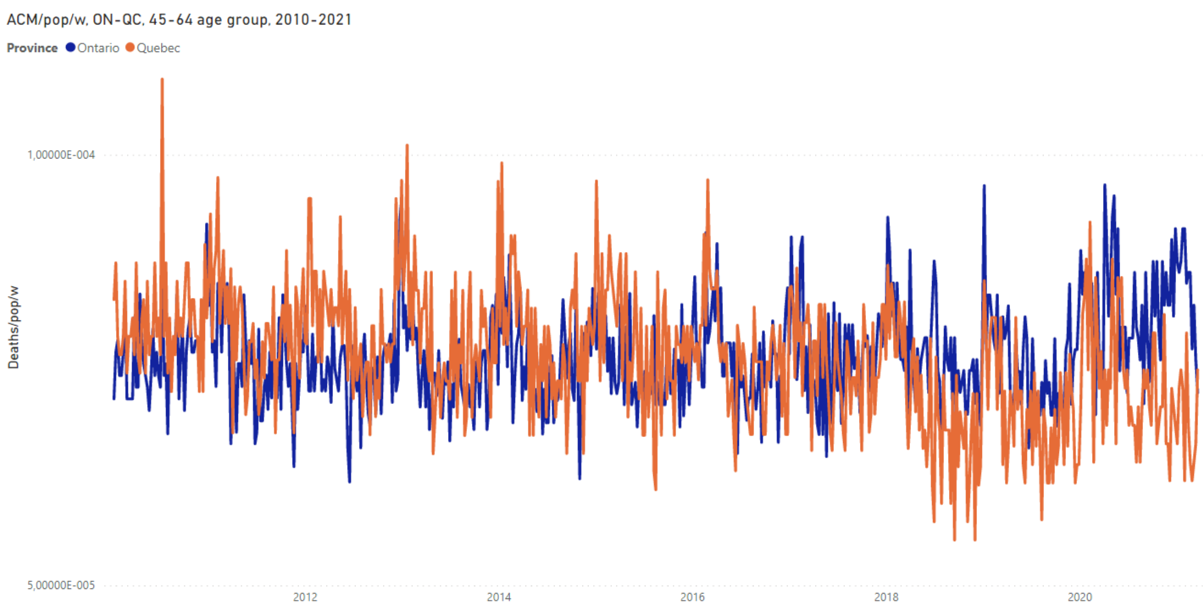


Figure A6: All-cause mortality by population by week in Ontario and Quebec for the 45-64 age group, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

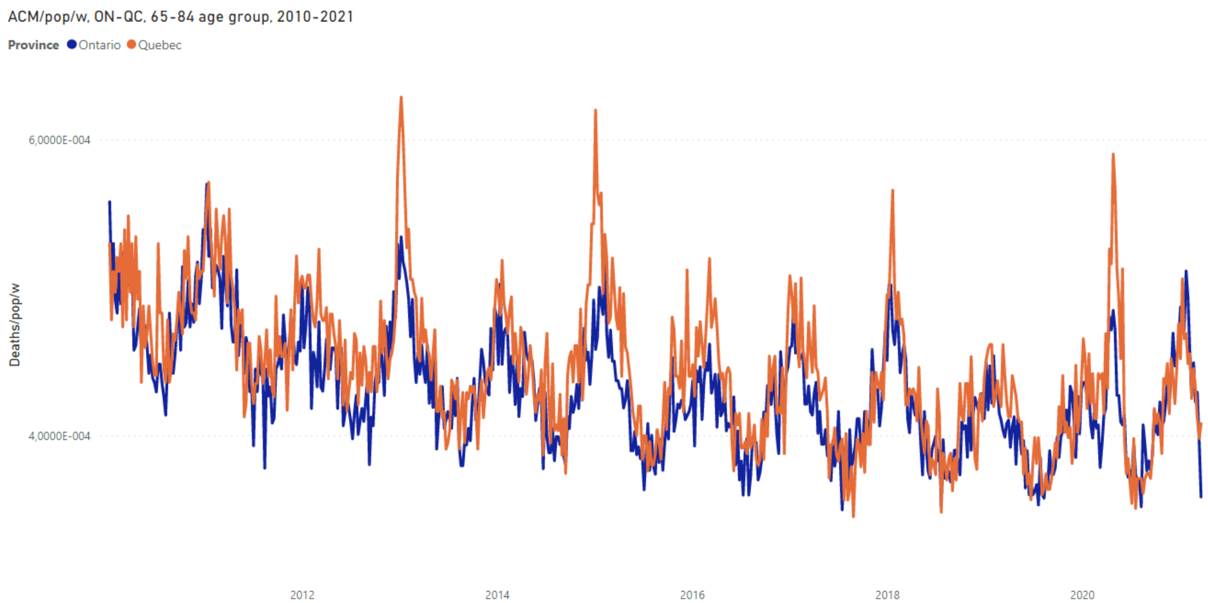


Figure A7: All-cause mortality by population by week in Ontario and Quebec for the 65-84 age group, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

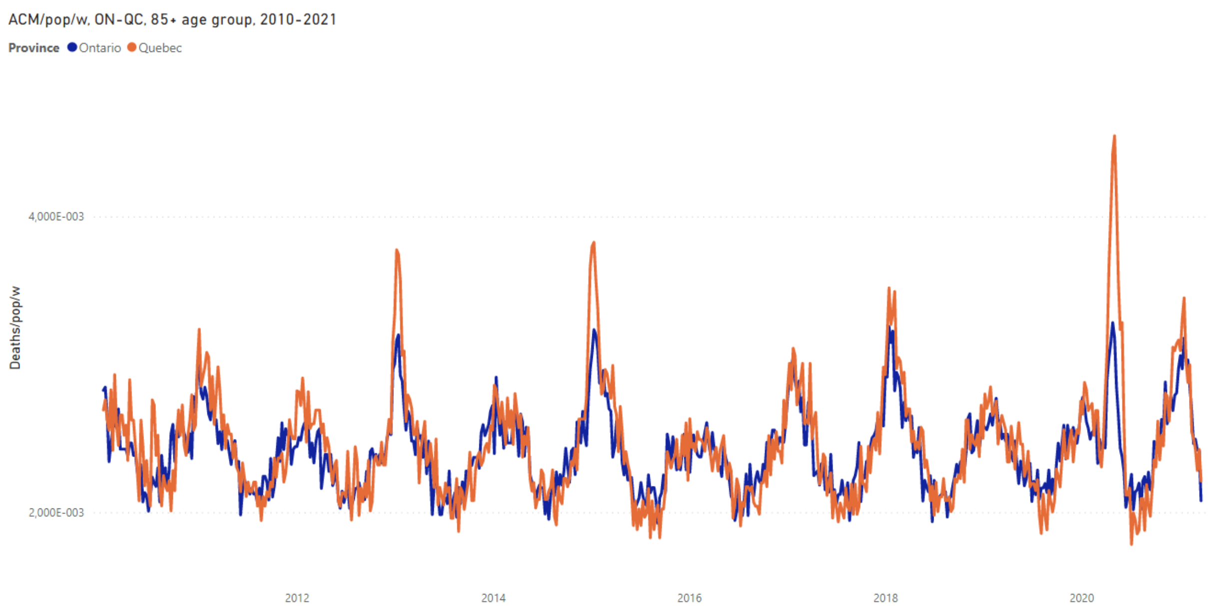


Figure A8: All-cause mortality by population by week in Ontario and Quebec for the 85+ age group, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

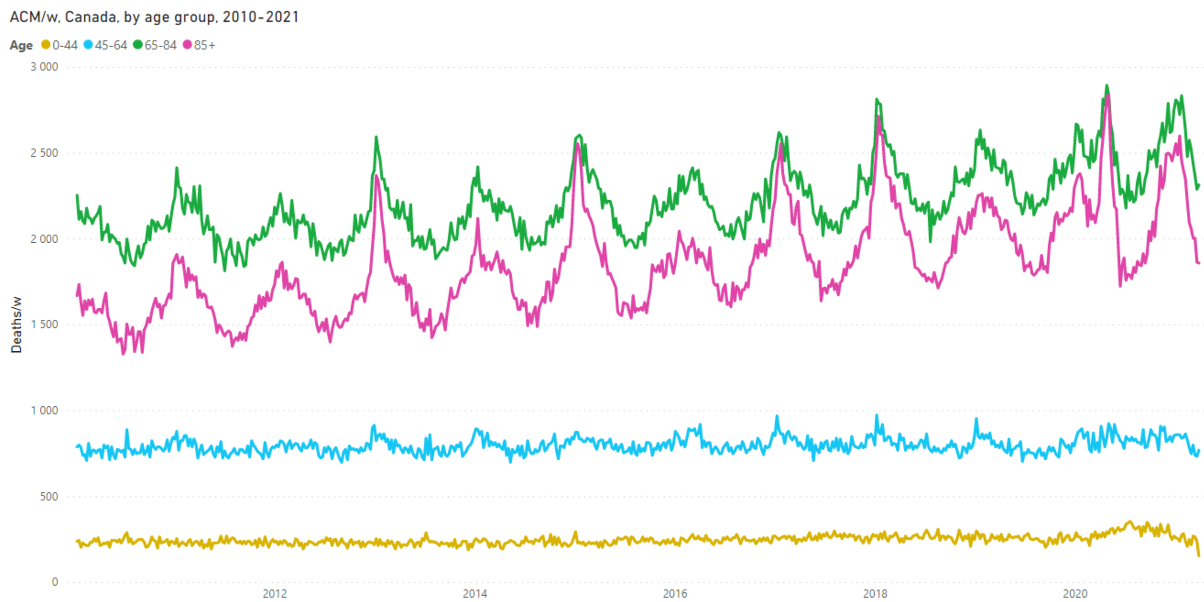


Figure A9: All-cause mortality by week in Canada by age group, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021).

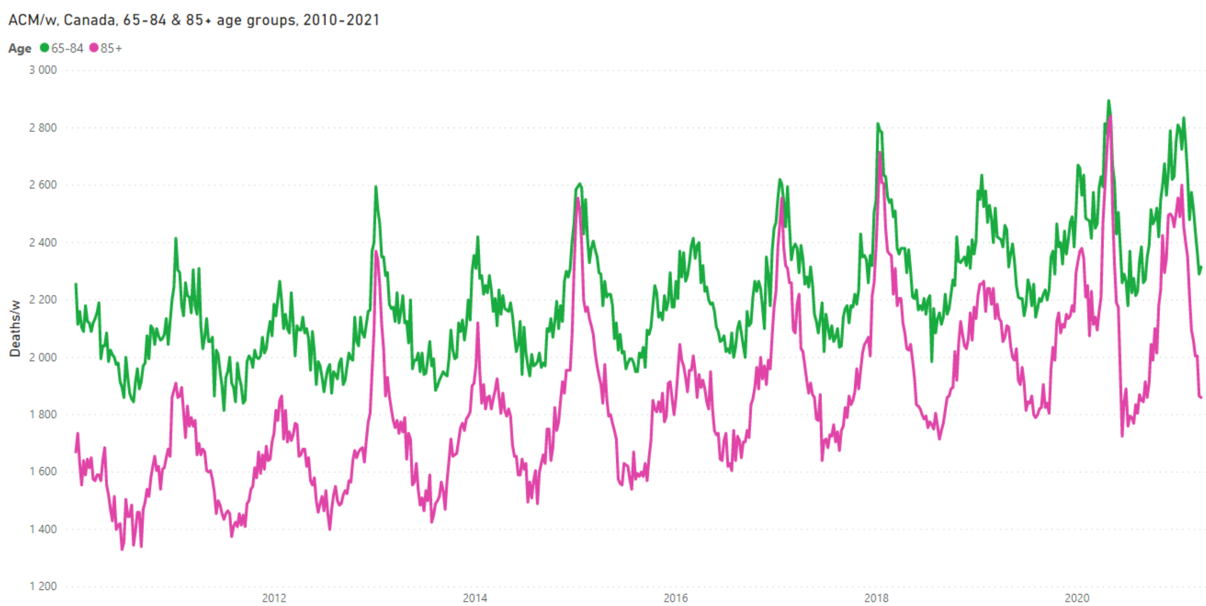


Figure A10: All-cause mortality by week in Canada for the 65-84 and 85+ age groups, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2021).

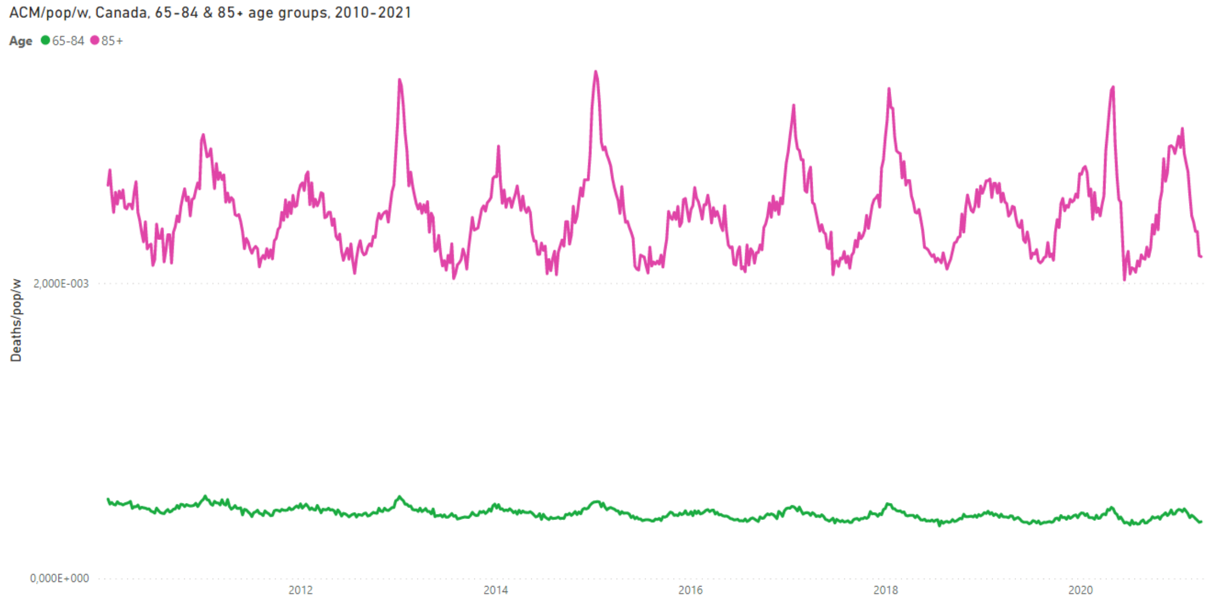


Figure A11: All-cause mortality by population by week in Canada for the 65-84 and 85+ age groups, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

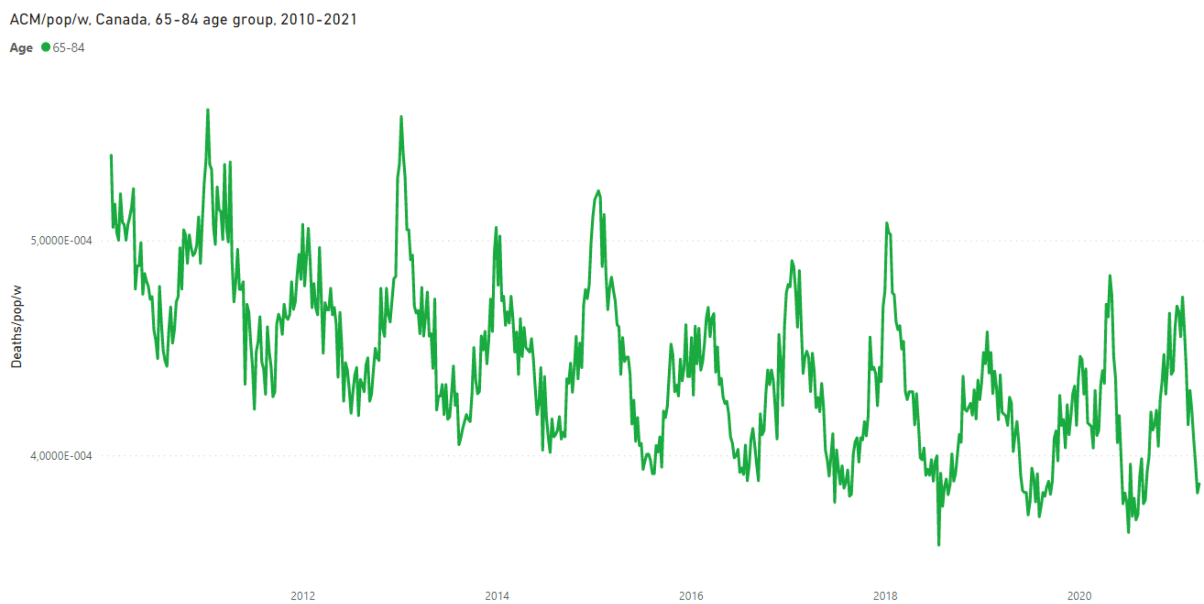


Figure A12: All-cause mortality by population by week in Canada for the 65-84 age group, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

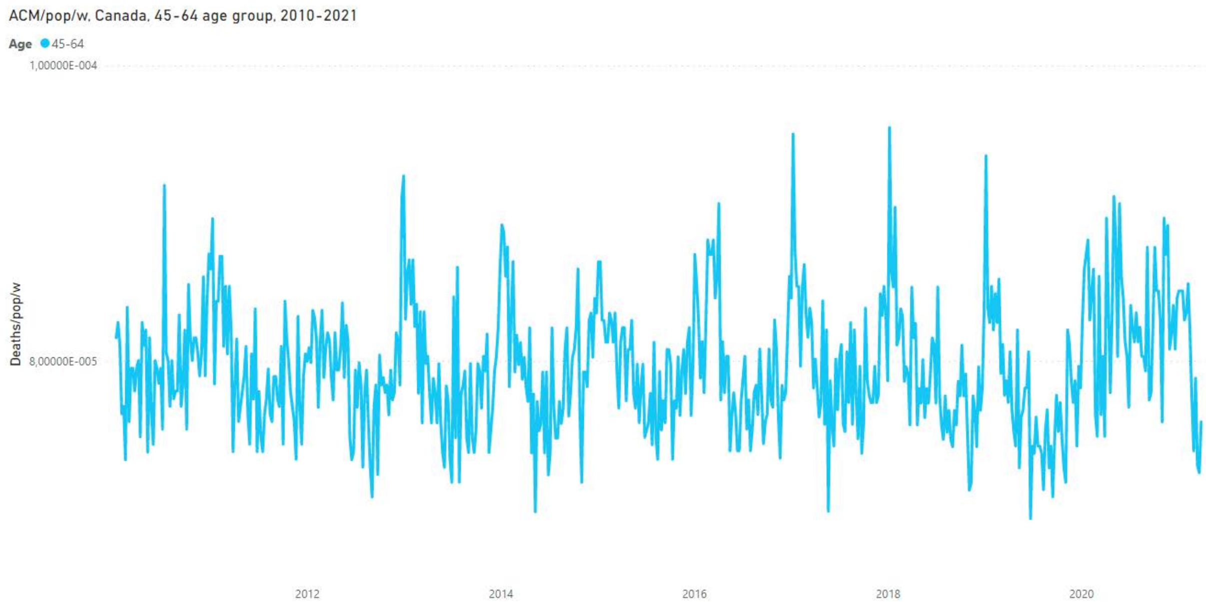


Figure A13: All-cause mortality by population by week in Canada for the 45-64 age group, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

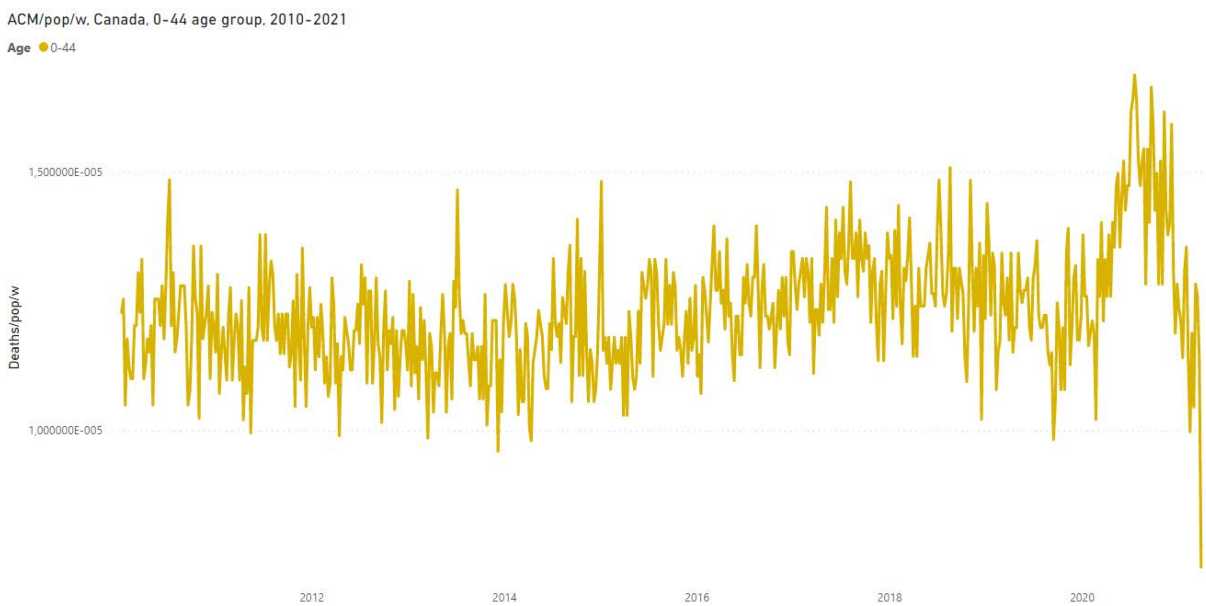


Figure A14: All-cause mortality by population by week in Canada for the 0-44 age group, both sexes, from 2010 to 2021. Data are displayed from January 2010 to March 2021. Data were retrieved from StatCan (StatCan, 2020 and 2021).

Review of scientific reports of harms caused by face masks, up to February 2021

Denis G. Rancourt, PhD

Researcher, Ontario Civil Liberties Association (ocla.ca)
Member scientist, PANDA (pandata.org)
[See section about the author's expertise, at the end]

Working report (not submitted for journal publication), published at Research Gate (https://www.researchgate.net/profile/D_Rancourt)

22 February 2021

The article is organized into the following sections:

- Summary
- Introduction: Government's onus to evaluate safety
- Context: Risk-benefit-harm analysis
- Healthcare workers (HCWs)
- Physiological impacts of face masks in healthy adults
- Psychological harm in the general population
- Infants and school children
- Microbial pathogen infections from masks
- Endnotes / References

Summary

It is a testimony to the power of propaganda, institutional capture, and the desire to socially conform that masking of the general population has successfully been imposed during the COVID-19 era. The harms from this imposition are palpable, and potentially long-term and gargantuan, not the least of which is the psychological training of the public to comply with an absurd measure that has direct personal negative impact. I review the mounting evidence of the obvious: Universal masking harms people and society, without any detectable benefit.

Introduction: Government's onus to evaluate safety

Following the precautionary principle, government has the onus to demonstrate absence of significant anticipated harm, prior to imposing a measure, especially with a personal medical measure applied to the general healthy population.

The precautionary principle was not followed for masks in the COVID-19 pandemic. The general masking implementations in Canadian provinces were even more aggressive than the qualified recommendations of the WHO [1].

This reckless government overreach has not been missed in recent scientific commentary. A few examples are as follows.

- As early as 20 April 2020, Lazzarino et al. directly opposed a logical perversion of the precautionary principle which has been applied by some scientists and many lawmakers (i.e., that governments should act "without definitive evidence, just in case"):

> "[W]hile no single formulation of that principle has been universally adopted,(ref) the precautionary principle aims at preventing researchers and policy makers from neglecting potentially-harmful side effects of interventions. […]
>
> Most scientific articles and guidelines in the context of the covid-19 pandemic highlight two potential side effects of wearing surgical face

masks in the public [false sense of security, inappropriate use of face mask], but we believe that there are other ones that are worth considering before any global public health policy is implemented involving billions of people. […]

[…] It is necessary to quantify the complex interactions that may well be operating between positive and negative effects of wearing surgical masks at population level. It is not time to act without evidence."

[2] 2020--Lazzarino : "Rapid Response: **Covid-19: important potential side effects of wearing face masks that we should bear in mind**". Antonio Lazzarino, A Steptoe, M Hamer, S Michie. 20 April 2020. *BMJ*. ---- https://www.bmj.com/content/369/bmj.m1435/rr-40

- On 13 August 2020, the surgeons Frountzas et al. warned that COVID-19 enthusiasm for imposing personal protective equipment (PPE) on surgeons could put surgery patients at risk (the equivalent can be said of train, tram, and bus drivers, and a large sector of workers servicing the public):

"Either in the case of a second lockdown or not, the safety of PPE use against COVID-19 for surgeons should be investigated. All parts of PPE increase surgeon's body temperature and sweating, leading to an impairment of surgeon's comfort, especially during prolonged and complicated surgical procedures. As mentioned above, PPE seems to be associated with important side effects, like dermatoses and headaches for healthcare workers. The PPE-associated discomfort and side effects during surgery may increase surgeons' anxiety and fatigue while performing difficult operations."

[3] 2020--Frountzas : M. Frountzas, C. Nikolaou, D. Schizas et al., "**Personal protective equipment against COVID-19: Vital for surgeons, harmful for patients?**", The American Journal of Surgery. 13 August 2020. https://doi.org/10.1016/j.amjsurg.2020.09.014

- By 22 November 2020, Dr. Vainshelboim was unambiguous:

"*Abstract:* … Although, scientific evidence supporting facemasks' efficacy is lacking, adverse physiological, psychological and health effects are established. Is has been hypothesized that facemasks have compromised safety and efficacy profile and should be avoided from use. The current article comprehensively summarizes scientific evidences with respect to wearing facemasks in the COVID-19 era. …

Long-Term health consequences of wearing facemasks: Long-term practice of wearing facemasks has strong potential for devastating health consequences. Prolonged hypoxic-hypercapnic state compromises normal physiological and psychological balance, deteriorating health and promotes the developing and progression of existing chronic diseases (10 refs).

Conclusion: … Wearing facemasks has been demonstrated to have substantial adverse physiological and psychological effects. These include hypoxia, hypercapnia, shortness of breath, increased acidity and toxicity, activation of fear and stress response, rise in stress hormones, immunosuppression, fatigue, headaches, decline in cognitive performance, predisposition for viral and infectious illnesses, chronic stress, anxiety and depression. Long-term consequences of wearing facemask can cause health deterioration, developing and progression of chronic diseases and premature death."

[4] 2021--Vainshelboim : Vainshelboim B. "**Facemasks in the COVID-19 era: A health hypothesis**". *Medical Hypotheses*. 2021;146:110411. doi:10.1016/j.mehy.2020.110411 ---- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7680614/

Indeed, harms from prolonged masking are increasingly being documented in many scientific studies, especially in the areas of healthcare workers, school children, newborn infants, and bacterial infections in the general population, as described below.

Context: Risk-benefit-harm analysis

In a broad policy perspective, three questions are relevant:

- What is the risk from COVID-19?
- Is there any evidence that face masks can reduce the risk from COVID-19?
- Do face masks cause harm?

Regarding the first question (What is the risk from COVID-19?), at this stage, almost a full calendar year since the pandemic was declared by the WHO on 11 March 2020, one

has an upper limit on the risk of dying from COVID-19 ("per year"), based on global statistics:

> Risk = number of deaths in a full yearly spread of the pandemic / population
>
> Risk < 2.43 M / 7.8 B = **0.03 %** (current WHO statistics, February 2021)

The thus calculated worldwide risk per year (0.03 %) is an overestimated upper bound because the deaths reported to the WHO by nation states are deaths "with" COVID-19, not deaths determined to be "caused by" COVID-19, and because the recommended RT-PRC test is not reliable, and because attribution of COVID-19 can be based on reported symptoms alone, without laboratory viral identification, in a global context of high likelihood of reporting bias.

More importantly, the thus calculated overestimated upper-bound risk (0.03 %) is further overestimated because it does not take into account the large and known age-dependent susceptibility for death from COVID-19. An age-susceptibility-corrected upper-bound risk can be estimated as follows. (The correction is needed because a COVID-19 death does not cause as many lost years lived as an average death from a cause that does not discriminate by age.)

- Global average age = 29.6 years
- Global life expectancy at birth = 71.5 years
- Global population = 7.8 B
- Global life-year pool = (7.8 B) x (71.5 - 29.6 years) = 327 B life-years
- Average loss of life years per COVID-19 death = 0.5 to 5 years, say 2.75 years
- Global loss of life-years from COVID-19 per year = (2.43 M per year) x (2.75 years) = 6.68 M life-years per year (of COVID-19 pandemic)
- Adjusted Risk < 6.68 M / 327 B = **0.002 %**

The unadjusted overestimated upper-bound global risk per year of dying from COVID-19 (0.03 %) is five times less than the risk per year of dying from cancer in Canada. The age-susceptibility-corrected (lost-life-years-adjusted) overestimated upper-bound risk per year from COVID-19 (0.002 %) is five times less than the risk per year of dying from a car accident in the USA.

Regarding the second question (Is there any evidence that face masks can reduce the risk from COVID-19?), as per [5] [6] [7]:

- The only way to scientifically measure the efficacy of masks is using a randomized controlled trial (RCT) with “verified outcome” (laboratory confirmed infection) because: (a) the efficacy is small compared to other known and unknown factors, (b) the person to person variations of infectiousness and susceptibility are known to be large compared to the averages, and (c) there is a high potential for bias in data collection/selection and in interpretation, in any substandard study.
- There have been no less than 15 policy-grade RCTs with verified outcome, in health care, community, and general-population settings. All but the most recent one have been analyzed in published formal systematic reviews. All 15 studies find that no reduction in risk of being infected can be detected with statistical significance. This means that any benefit is too small to be detected by science.
- The government claims that masks work are in effect disingenuous propaganda, improperly relying on substandard and irrelevant studies (Exhibit-54).
- Therefore, the presumption that masks work is incorrect. It is disproved by science: Any risk reduction is too small to be detected using usual and established statistical criteria.

There is no reliable or policy-grade evidence that face masks can reduce the risk from COVID-19.

Regarding the third question (Do face masks cause harm?), as indicated above, there is presently a surge of scientific reports about harm caused by face masks, which I describe below.

There is no doubt that prolonged mask wearing causes significant harm and disability to healthy individuals. Recent studies have focussed on:

- healthcare workers
- school children
- newborn infants
- healthy adults

The early review (19 June 2020) of Bakhit et al. was for harms from face masks in any setting (home, workplace, etc.). They screened 5471 potential articles and identified 37 studies that reliably reported harms from masks. These 37 studies were published as early as 2004, and included two studies published in 2020. In these 37 studies (their Table 1): 20 reported “discomfort and irritation”; 4 reported “dyspnoea & other”; 6 reported “psychological impacts”; 9 reported “communication impacts”; and “mask contamination” was reported in one study. Bakhit et al.’s Conclusion (in Abstract) was:

"There are insufficient data to quantify all of the adverse effects that might reduce the acceptability, adherence, and effectiveness of face masks. New research on facemasks should assess and report the harms and downsides. Urgent research is also needed on methods and designs to mitigate the downsides of facemask wearing, particularly the assessment of alternatives such as face shields."

[8] 2020--Bakhit : "**Downsides of face masks and possible mitigation strategies: a systematic review and meta-analysis**". Mina Bakhit, Natalia Krzyzaniak, Anna Mae Scott, Justin Clark, Paul Glasziou, Chris Del Mar. *medRxiv* 2020.06.16.20133207; doi: https://doi.org/10.1101/2020.06.16.20133207. Now accepted for publication in *BMJ* Open. ---- https://www.medrxiv.org/content/10.1101/2020.06.16.20133207v1

Healthcare workers (HCWs)

Not eight months later, following the Bakhit et al. review, Galanis et al. (5 February 2021, preprint) published a systematic review and meta-analysis to "assess the impact of PPE use on HCWs' physical health during the COVID19 pandemic". Their "review included 14 studies with 11746 HCWs from 16 counties":

[9] 2021--Galanis : Galanis P, Vraka I, Fragkou D, Bilali A, Kaitelidou D. "**Impact of personal protective equipment use on health care workers' physical health during the COVID-19 pandemic: a systematic review and meta-analysis**". *medRxiv*; 2021. DOI: 10.1101/2021.02.03.21251056. ---- https://www.medrxiv.org/content/medrxiv/early/2021/02/05/2021.02.03.21251056.full.pdf

Nine prominent recent studies focussed on healthcare workers (HCWs) are as follows:

→ "*Results (Abstract):* A total of 343 healthcare professionals on the COVID-19 front lines participated in this study [New York City]. 314 respondents reported adverse effects from prolonged mask use with headaches being the most common complaint (n = 245). Skin breakdown was experienced by 175 respondents, and acne was reported in 182 respondents.

Impaired cognition was reported in 81 respondents. … Some respondents experienced resolved side effects once masks were removed, while others required physical or medical intervention.

Conclusion (Abstract): Prolonged use of N95 and surgical masks by healthcare professionals during COVID-19 has caused adverse effects such as headaches, rash, acne, skin breakdown, and impaired cognition in the majority of those surveyed. …"

[10] 2020--Rosner : Elisheva Rosner E (2020) "**Adverse Effects of Prolonged Mask Use among Healthcare Professionals during COVID-19**". *Journal of Infectious Disease and Epidemiology* 6:130. doi.org/10.23937/2474-3658/1510130 ---- https://clinmedjournals.org/articles/jide/journal-of-infectious-diseases-and-epidemiology-jide-6-130.php

→ "*Abstract:* … All participants wore either surgical masks or N95 respirators for a minimum of 4 h per day [India]. … A total of 250 healthcare workers participated in the study … The acquired results were excessive sweating around the mouth accounting to 67.6%, difficulty in breathing on exertion 58.2%, acne 56.0% and itchy nose 52.0%. This study suggests that prolonged use of facemasks induces difficulty in breathing on exertion and excessive sweating around the mouth to the healthcare workers which results in poorer adherence and increased risk of susceptibility to infection."

[11] 2021--Purushothaman : Purushothaman, P.K., Priyangha, E. & Vaidhyswaran, R. "**Effects of Prolonged Use of Facemask on Healthcare Workers in Tertiary Care Hospital During COVID-19 Pandemic**". *Indian J Otolaryngol Head Neck Surg* 73, 59–65 (2021). https://doi.org/10.1007/s12070-020-02124-0

→ "*Results (Abstract):* A total of 158 healthcare workers participated in the study [Singapore]. … Out of 158 respondents, 128 (81.0%) respondents developed de novo PPE-associated headaches. A pre-existing primary headache diagnosis (OR = 4.20, 95% CI 1.48-15.40; P = .030) and combined PPE usage for >4 hours per day (OR 3.91, 95% CI 1.35-11.31; P = .012) were independently associated with de novo PPE-associated headaches. Since COVID-19 outbreak, 42/46 (91.3%) of respondents with pre-existing headache diagnosis either "agreed" or "strongly agreed" that the increased PPE usage had affected the control of their background headaches, which affected their level of work performance.

Conclusion (Abstract): Most healthcare workers develop de novo PPE-associated headaches or exacerbation of their pre-existing headache disorders."

[12] 2020--Ong : Ong JJY, Bharatendu C, Goh Y, Tang JZY, Sooi KWX, Tan YL, Tan BYQ, Teoh HL, Ong ST, Allen DM, Sharma VK. "**Headaches Associated With Personal Protective**

Equipment - A Cross-Sectional Study Among Frontline Healthcare Workers During COVID-19". *Headache: The Journal of Head and Face Pain*. 2020 May;60(5):864-877. doi: 10.1111/head.13811. Epub 2020 Apr 12. PMID: 32232837. ---- https://pubmed.ncbi.nlm.nih.gov/32232837/

[13] 2020--Magnavita (critique of Ong, 2020): Magnavita, N. and Chirico, F. (2020), "**Headaches, Personal Protective Equipment, and Psychosocial Factors Associated With COVID-19 Pandemic**". *Headache: The Journal of Head and Face Pain*, 60: 1444-1445. https://doi.org/10.1111/head.13882

[14] 2020--Goh (response to critique of Ong, 2020): Goh Y, Ong JJY, Bharatendu C, Tan BYQ, Sharma VK. "**Headaches Due to Personal Protective Equipment During COVID-19 Pandemic: A Comment**". *Headache: The Journal of Head and Face Pain*. 2020;60(7):1446-1447. doi:10.1111/head.13879 ---- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7323331/

→ "*Results (Abstract):* A total of 400 healthcare providers completed the questionnaire, 383 of them met the inclusion criteria [Italy]. The majority were doctors, with a mean age of 33.4 ± 9.2 years old. Among 166/383 subjects, who were headache free at baseline, 44 (26.5%) developed de novo headache. Furthermore, 217/383 reported a previous diagnosis of primary headache disorder: 137 were affected by migraine and 80 had tension-type headache. A proportion (31.3%) of these primary headache sufferers experienced worsening of their pre-existing headache disorder, mainly for migraine frequency and attack mean duration.

Conclusions (Abstract): Our data showed the appearance of de novo associated facemask headache in previous headache-free subjects and an exacerbation of pre-existing primary headache disorders, mostly experienced by people with migraine disease."

[15] 2021--Rapisarda : Rapisarda, L., Trimboli, M., Fortunato, F. et al. "**Facemask headache: a new nosographic entity among healthcare providers in COVID-19 era**". *Neurological Sciences* (2021). https://doi.org/10.1007/s10072-021-05075-8

→ "*Conclusion (Abstract):* (A total of 155 healthcare workers responded to the questionnaire [Morocco].) The increased use of PPE, especially high filtrating masks during the COVID-19 outbreak is responsible for generating headaches in healthcare workers on frontline (62%) either De novo (33%) or as an aggravation of pre-existing one (29%). Working conditions have the greater impact on generating these types of headaches more than any pre-existing comorbidity. …"

[16] 2020--Hajjij : Hajjij A, Aasfara J, Khalis M, et al. "**Personal Protective Equipment and Headaches: Cross-Sectional Study Among Moroccan Healthcare Workers During COVID-**

19 Pandemic". *Cureus*. 2020 Dec;12(12):e12047. DOI: 10.7759/cureus.12047. ----
https://europepmc.org/article/med/33447477

→ *Results (Abstract):* (315 participants, Turkey) … New-onset symptom rate was 66% (n=208). The most common new-onset symptom was headache (n=115, 36.5%) followed by breathing difficulty-palpitation (n=79, 25.1%) and dermatitis (n=64, 20.3%). Extended use of PPE, smoking, and overweight were independently associated with developing new-onset symptoms. A clear majority of symptomatic participants pointed out impact on working performance (193/208, 92.7%).

[17] 2020-- Çağlar : Çağlar, A., Kaçer, İ, Hacımustafaoğlu, M., Öztürk, B., & Öztürk, K. (2020). "**Symptoms associated with personal protective equipment among frontline healthcare professionals during the COVID-19 pandemic**". *Disaster Medicine and Public Health Preparedness*, 1-15. doi:10.1017/dmp.2020.455 ----
https://www.cambridge.org/core/journals/disaster-medicine-and-public-health-preparedness/article/symptoms-associated-with-personal-protective-equipment-among-frontline-healthcare-professionals-during-the-covid19-pandemic/FD3DF0B1437D8E4C9C577D09A2295C68

→ "*Results (Abstract):* The subjects are n=306, 244 women (79.7%), with an average age of 43 years (range 23–65) [Spain]. Of the total, 129 (42.2%) were physicians, 112 (36.6%) nurses and 65 (21.2%) other health workers. 208 (79.7%) used surgical masks and 53 (20.3%) used filter masks. Of all those surveyed, 158 (51.6%) presented 'de novo' headache. The occurrence of a headache was independently associated with the use of a filter mask, OR 2.14 (95% CI 1.07 to 4.32); being a nurse, OR 2.09 (95% CI 1.18 to 3.72) or another health worker, OR 6.94 (95% CI 3.01 to 16.04); or having a history of asthma, OR 0.29 (95% CI 0.09 to 0.89). According to the type of mask used, there were differences in headache intensity, and the impact of a headache in the subjects who used a filter mask was worse in all the aspects evaluated.

Conclusion (Abstract): The appearance of 'de novo' headache is associated with the use of filter masks and is more frequent in certain healthcare workers, causing a greater occupational, family, personal and social impact."

[18] 2020--Ramirez-Moreno : Ramirez-Moreno JM, Ceberino D, Gonzalez Plata A, et al. "**Mask-associated 'de novo' headache in healthcare workers during the COVID-19 pandemic**". *Occupational and Environmental Medicine*. Published Online First: 30 December 2020. doi: 10.1136/oemed-2020-106956 ---- https://oem.bmj.com/content/early/2020/12/29/oemed-2020-106956

→ "*Results:* … Out of 241 [Pakistan], 68 participants (28.2%) reported de novo headaches since the start of the pandemic, with majority describing the headache as bilateral in location (n = 47, 69%), with pressure/heaviness in quality (n = 31, 45.5%) and moderate in intensity (n = 45, 66%). … Out of the 68 participants with new-onset headaches, 16 (23.5%) stated that the headache started more than 2 hours after donning PPE, while 19 (27.9%) participants stated that the headache ended between 1-2 hours after doffing of PPE. Fifty-three respondents (77.9%) experienced the headaches for 4 or less days per month. …"

[19] 2020--Zaheer : Rumeesha Zaheer, Maheen Khan, Ahmed Tanveer, Amal Farooq, Zohaib Khurshid. "**Association of Personal Protective Equipment with De Novo Headaches In Frontline Healthcare Workers during COVID-19 Pandemic: A Cross-Sectional Study**". *European Journal of Dentistry*. 2020 Dec;14(S 01):S79-S85. doi: 10.1055/s-0040-1721904. Epub 2020 Dec 26. PMID: 33368069; PMCID: PMC7775222. ---- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7775222/

→ "…Several dermatoses [skin defects or lesions on the skin] have been reported due to PPE, such as pressure injury, contact dermatitis, pressure urticaria [hives] and exacerbation of pre-existing skin diseases, including seborrheic dermatitis [scales] and acne.(2 refs) We report a preliminary data of HCW who experienced facial dermatoses due to the use of PPE. From 24 March 2020 to 16 April 2020, we came across with 43 patients comprising physicians, nurses and paramedical staff who involved (directly/indirectly) in managing patients of COVID-19 [India]. … The most commonly noted dermatoses were irritant contact dermatitis (ICD; 39.5%) followed by friction dermatitis (25.5%). Goggles were the most common culprit agent among all PPE causing any one of the dermatoses (51.92%), followed by N95 masks (30.77%) and face shields (17.31%). Nasal bridge (63%) was the commonest anatomical site affected due to dermatoses followed by cheeks and chin (26%). However, there was a considerable overlap of different dermatoses with affliction of multiple sites. The most common symptom experienced by patients was pruritus [itchiness] (67.44%), while erythema [redness] (53.49%) was the most common sign observed. Interestingly, we observed two distinct dermatoses, i.e. whole face erythema (suffusion; 21%) attributed to doffing after a long shift and lip lick dermatitis due to constant licking of lips, because of feeling of intense thirst due to restricted fluid intake after donning PPE. The duration of wearing the goggles and mask, excessive sweating and ill-fitting masks, all were associated with increased sensation of irritation. Most of these dermatoses responded well to topical moisturizer, calamine lotion and oral antihistamines. Overall, 21% patients suffered from work absenteeism due to one of the dermatoses. Personal protective equipment-induced dermatoses occur mainly due to the occlusion and hyper-hydration effect of PPE and friction leading breach in the epidermal integrity.(ref) Recently, in China, authors noted a very high prevalence, i.e. 97% of skin damages in first-line HCW fighting COVID-19.(ref)"

[20] 2020--Singh : Singh, M., Pawar, M., Bothra, A., Maheshwari, A., Dubey, V., Tiwari, A. and Kelati, A. (2020), "**Personal protective equipment induced facial dermatoses in healthcare workers managing Coronavirus disease 2019**". *Journal of the European Academy of Dermatology and Venereology*, 34: e378-e380. https://doi.org/10.1111/jdv.16628

Physiological impacts of face masks in healthy adults

In addition to the large focus on healthcare workers, a significant body of recent studies is accumulating about the harms to infants and school children (described below). Also, studies about measured physiological impacts of face masks in healthy adults are beginning. In 2005, Li et al. reported on the temperature and humidity microclimates of face masks; and apparently the first physiological measurements on masked healthy adults were reported in 2020 by Fikenzer et al.:

→ "*Discussion (Abstract):* We discuss how N95 and surgical facemasks induce significantly different temperature and humidity in the microclimates of the facemasks, which have profound influences on heart rate and thermal stress and subjective perception of discomfort."

[21] 2005--Li : Li Y, Tokura H, Guo YP, et al. "**Effects of wearing N95 and surgical facemasks on heart rate, thermal stress and subjective sensations**". *Int Arch Occup Environ Health*. 2005;78(6):501-509. doi:10.1007/s00420-004-0584-4 ---- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7087880/

→ "*Discussion:* This first randomized cross-over study assessing the effects of surgical masks and FFP2/N95 masks on cardiopulmonary exercise capacity yields clear results. Both masks have a marked negative impact on exercise parameters such as maximum power output (Pmax) and the maximum oxygen uptake (VO2max/kg). FFP2/N95 masks show consistently more pronounced negative effects compared to surgical masks. Both masks significantly reduce pulmonary parameters at rest (FVC, FEV1, PEF) and at maximum load (VE, BF, TV). …

Pulmonary function: … The data of this study are obtained in healthy young volunteers, the impairment is likely to be significantly greater, e.g., in patients with obstructive pulmonary diseases (ref). From our data, we conclude that wearing a medical face mask has a significant impact on pulmonary parameters both at rest and during maximal exercise in healthy adults.

Cardiac function: … These data suggest a myocardial [relating to the muscular tissue of the heart] compensation for the pulmonary limitation in the healthy volunteers. In patients with impaired myocardial function, this compensation may not be possible."

[22] 2020--Fikenzer : Fikenzer S, Uhe T, Lavall D, Rudolph U, Falz R, Busse M, Hepp P, Laufs U. "**Effects of surgical and FFP2/N95 face masks on cardiopulmonary exercise capacity**". *Clin Res Cardiol.* 2020 Dec;109(12):1522-1530. doi: 10.1007/s00392-020-01704-y. Epub 2020 Jul 6. PMID: 32632523; PMCID: PMC7338098. - https://link.springer.com/article/10.1007/s00392-020-01704-y

Psychological harm in the general population

One research focus area that appears to be entirely lacking, in examining the harms of masks, is the broad psychological (and therefore social) impact of mandatory masking policies applied to the general population.

The current knowledge of the individual's fundamental psychological needs that determine well-being is expressed in the modern theory known as "self-determination theory" (SDT), which is also the scientific basis for personal motivation:

> "Self-Determination Theory (SDT) proposes that certain evolved psychological needs must be satisfied if individuals are to develop to their fullest potential, in the same way that plants require key nutrients to thrive (refs). SDT posits three universal needs: autonomy, competence, and relatedness. Autonomy involves the need to experience one's behavior as freely chosen and volitional, rather than imposed by external forces. Competence involves the need to feel capable and effective in one's actions. Relatedness involves the need for belonging, intimacy, and connectedness to others. SDT theorists view these needs as broad motivational tendencies that operate across life domains and contend that satisfaction of all three needs, not just one or two, is essential for well-being. Although the expression or means of satisfying these needs may vary across cultures, their satisfaction is viewed as essential for well-being in all cultures." [highlights added]
>
> [23] 2013--Church : Church AT, Katigbak MS, Locke KD, et al. "**Need Satisfaction and Well-Being: Testing Self-Determination Theory in Eight Cultures**". *Journal of Cross-Cultural Psychology*. 2013;44(4):507-534. doi:10.1177/0022022112466590 ----

https://www.webpages.uidaho.edu/klocke/publications/2013%20Church%20etal%20JCCP.pdf

There can be little doubt that forced masking of the general population has a significant potential to deteriorate the three fundamental psychological needs of the individual: autonomy, competence, and relatedness. This harm to individuals and the societal implications have not been studied. The impact may be gargantuan.

Only infants and school children have so far been considered using the perspective of psychological and developmental impact (as described below).

The 11 August 2020 Commentary of Scheid et al. is not helpful, because it incorrectly disregards physiological impacts and examines psychology solely from the perspective of mask compliance [24]. (Scheid JL, Lupien SP, Ford GS, West SL. "Commentary: Physiological and Psychological Impact of Face Mask Usage during the COVID-19 Pandemic". *Int J Environ Res Public Health*. 2020 Sep 12;17(18):6655. doi: 10.3390/ijerph17186655. PMID: 32932652; PMCID: PMC7558090. ---- https://pubmed.ncbi.nlm.nih.gov/32932652/)

Infants and school children

When considering whether a world of masked adults and children, at a crucial period in a baby's or child's life, can have long-term detrimental psychological and development impact, I propose that the following hierarchical sequence of thought experiments is useful:

- Would babies and children entirely raised by mechanical robots be adversely affected?
- Would babies and children entirely raised by masked adults, and themselves forced to be masked beyond two years of age, be adversely affected?
- What periods, durations and circumstances of masking, distancing and shielding could have long-term psychological or developmental negative consequences?

Given the known large impact that government measures have had on school children worldwide (see below), it should be of concern to us all that apparently the first scientific

analysis to consider risk-benefit analysis for school children was published as late as August 2020. On 6 August 2020, Spitzer submitted several central propositions:

→ "*Abstract:* … covering the lower half of the face reduces the ability to communicate, interpret, and mimic the expressions of those with whom we interact. Positive emotions become less recognizable, and negative emotions are amplified. Emotional mimicry, contagion, and emotionality in general are reduced and (thereby) bonding between teachers and learners, group cohesion, and learning – of which emotions are a major driver.

1. Introduction: … along with other measures of physical distancing and economic lockdowns, school closures were implemented during March 2020 affecting more than 1.5 billion students (children and adolescents) around the globe (ref). These closures of schools lasted for a few weeks only (as in Denmark) up to several months (in Italy and many other countries; (ref)) and led to marked decreases in educational gains (ref), hunger (because school meals were no longer served), increases in child abuse (because children were no longer observed by school staff), and, in general, the risk of "scarring the life chances of a generation of young people"(ref) (because of the long-term psychological, physiological, educational and even economic burden (ref), that societies put on their most vulnerable members; (ref))…

• … wearing masks may have physical side effects.
• Face masks impair face recognition and face identification.
• Face masks impair verbal and non-verbal communication.
• Face masks block emotional signaling between teacher and learner.

Given these pros and cons, it is not clear whether face masks should play a major role in educational settings in times of the current viral pandemic. … This matter should be discussed urgently, since it globally affects more than 1.5 billion students, teachers, and school staff directly, and, in addition, their families indirectly.

6. Face masks block emotional signaling between teachers and students: … In sum, recognition of, and response to, the outward emotional displays of one's peers' faces is a critical and necessary component of social interaction in schools. It helps pupils and teachers to modify their behavior in order to align with social communication and behavioral norms. When these emotional displays are inhibited by face masks, our ability to communicate effectively with one another is reduced and we are primarily left with mimicking negative (frown) emotions. All of this happens primarily outside of conscious awareness, and hence, is hard to be consciously controlled or even corrected. Since emotions are a major driver of group cohesion, the decreased emotionality, and decreased positive emotionality in particular, may interfere with smooth classroom action. Given the fact that the very process of learning is facilitated by emotions (this is their main raison d´être), face masks are likely to cause some interference with pedagogy." [highlights are added]

[25] 2020--Spitzer : Spitzer M. "**Masked education? The benefits and burdens of wearing face masks in schools during the current Corona pandemic**". *Trends in Neuroscience and*

Education. 2020;20:100138. doi:10.1016/j.tine.2020.100138 ---- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7417296/

Still later, two studies pointed out the likelihood that babies are significantly harmed by general masking practices. Especially, the 22 September 2020 study of Green et al. alerted us to "potential negative effects of masks on long-term development related to human connection and attachment":

→ "*Abstract:* … COVID-19 has changed the way that newborn babies are cared for within the neonatal setting due to the introduction of social distancing and wearing of face masks to limit the spread of the infection. Potential implications exist related to the normal development of bonding and connections with others. This paper discusses the importance of face to face interactions for early attachment between babies and parents within the context of relevant underpinning developmental theory. …"

[26] 2021--Green : Green, Janet et al. "**The implications of face masks for babies and families during the COVID-19 pandemic: A discussion paper**". *Journal of neonatal nursing* : JNN vol. 27,1 (2021): 21-25. doi:10.1016/j.jnn.2020.10.005 - https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7598570/

Likewise, on 11 February 2021, Lewkowicz pointed out the following about language acquisition by babies:

→ "…the COVID pandemic has laid bare our fundamental need to see whole faces. Could it be that babies and young children, who must learn the meaning of the myriad communicative signals normally available in their social partners' faces, are especially vulnerable to their degradation in partially visible faces? … in my lab … We discovered that babies begin lip-reading at around 8 months of age. … Crucially, once lip-reading emerges in infancy, it becomes the default mode of speech processing whenever comprehension is difficult. …

Overall, the research to date demonstrates that the visible articulations that babies normally see when others are talking play a key role in their acquisition of communication skills. Research also shows that babies who lip-read more have better language skills when they're older. If so, this suggests that masks probably hinder babies' acquisition of speech and language."

[27] 2021--Lewkowicz : "**Masks Can Be Detrimental to Babies' Speech and Language Development**". David J. Lewkowic. *Scientific American*. Cogntion, Opinion. 11 February 2021. -

--- https://www.scientificamerican.com/article/masks-can-be-detrimental-to-babies-speech-and-language-development/

On 20 August 2020, Karvounides et al. submitted that mask wearing is a potential trigger for youth with chronic migraine:

→ “Many common triggers such as dehydration, fasting, sleep problems, and stressors were discussed above. Here we highlight [computer] screen use and mask wearing as potential additional school-related triggers. … Pressure created by the mask or its straps against various contact points on the face or scalp could trigger headache”

[28] 2021--Karvounides : Karvounides, D., Marzouk, M., Ross, A.C., VanderPluym, J.H., Pettet, C., Ladak, A., Ziplow, J., Patterson Gentile, C., Turner, S., Anto, M., Barmherzig, R., Chadehumbe, M., Kalkbrenner, J., Malavolta, C.P., Clementi, M.A., Gerson, T. and Szperka, C.L. (2021), “**The intersection of COVID-19, school, and headaches: Problems and solutions**”. *Headache: The Journal of Head and Face Pain*, 61: 190-201. https://doi.org/10.1111/head.14038

The idea of a mask is to breathe through the material and not have large gaps. This implies fastening bands and a tight fit, which implies pressure on the head, ears, nose, and face. The pressure points, in turn cause discomfort, at the very least, which is aggravated by lengthy duration and micro-environmental, psychological and physiological effects. Removing the pressure or the mouth and nose coverage defeats the purpose of the mask, in the belief that masks work to prevent transmission of the virus. And there are always unforeseen negative effects, such as causing permanent ear protrusion:

→ “Abstract: … Among those on the market, surgical masks with elastic loops are the ones most chosen by parents for their children. These elastics cause constant compression on the skin and, consequently, on the cartilage of the auricle, leading to erythematous and painful lesions of the retroauricular skin when the masks are used for many hours a day. Pre-adolescent children have undeveloped auricular cartilage with less resistance to deformation; prolonged pressure from the elastic loops of the mask at the hollow or, even worse, at the anthelix level can influence the correct growth and angulation of the outer ear. In fact, unlike when using conservative methods for the treatment of protruding ears, this prolonged pressure can increase the cephaloauricular angle of the outer auricle. It is important for the authorities

supplying the masks to be aware of this potential risk and for alternative solutions to be found …"

[29] 2020--Zanotti : Zanotti, B., Parodi, P.C., Riccio, M. et al. "**Can the Elastic of Surgical Face Masks Stimulate Ear Protrusion in Children?**". *Aesth Plast Surg* 44, 1947–1950 (2020). https://doi.org/10.1007/s00266-020-01833-9 - https://link.springer.com/article/10.1007/s00266-020-01833-9

Most importantly, however, whereas most professional public health agents and health researchers have been loath to embark on objective risk-benefit analysis, parents in Germany have answered a recent research-group's call to provide observations regarding masks on children. On 18 December 2020, Schwarz et al. reported striking results. Here is the full (v2) abstract of their preprint:

→ "*ABSTRACT*

Background: Narratives about complaints in children and adolescents caused by wearing a mask are accumulating. There is, to date, no registry for side effects of masks.

Methods: At the University of Witten/Herdecke an online registry has been set up where parents, doctors, pedagogues and others can enter their observations. On 20.10.2020, 363 doctors were asked to make entries and to make parents and teachers aware of the registry.

Results: By 26.10.2020 the registry had been used by 20,353 people. In this publication we report the results from the parents, who entered data on a total of 25,930 children. The average wearing time of the mask was 270 minutes per day. Impairments caused by wearing the mask were reported by 68% of the parents. These included irritability (60%), headache (53%), difficulty concentrating (50%), less happiness (49%), reluctance to go to school/kindergarten (44%), malaise (42%) impaired learning (38%) and drowsiness or fatigue (37%).

Discussion: This world's first registry for recording the effects of wearing masks in children is dedicated to a new research question. Bias with respect to preferential documentation of children who are particularly severely affected or who are fundamentally critical of protective measures cannot be dismissed. The frequency of the registry's use and the spectrum of symptoms registryed indicate the importance of the topic and call for representative surveys, randomized controlled trials with various masks and a renewed risk-benefit assessment for the vulnerable group of children: adults need to collectively reflect the circumstances under which they would be willing to take a residual risk upon themselves in favor of enabling children to have a higher quality of life without having to wear a mask."

[30] 2021--Schwarz : Silke Schwarz, Ekkehart Jenetzky, Hanno Krafft, Tobias Maurer, David Martin. “**Corona children studies "Co-Ki": First results of a Germany-wide registry on mouth and nose covering (mask) in children**”. 18 December 2020. DOI: 10.21203/rs.3.rs-124394/v1 - https://www.researchsquare.com/article/rs-124394/v1 ---- v2 (5 January 2021): https://www.researchsquare.com/article/rs-124394/v2

Microbial pathogen infections from masks

Finally, regarding potential mask harms, a notoriously understudied aspect is the potential population and individual health impacts of the development of bacterial and other pathogens on warm and humid cloth masks [1] [5] [7]. Matuschek et al. briefly reported it this way, without reference or demonstration:

→ “If masks are not exchanged regularly (or washed properly when made of cloth), pathogens can accumulate in the mask. When improperly used, the risk of spreading the pathogen—including SARS-CoV-2—might be critically increased.” (p. 5)

[31] 2020--Matuschek : Matuschek, C., Moll, F., Fangerau, H. et al. “**Face masks: benefits and risks during the COVID-19 crisis**”. *European Journal of Medical Research* 25, 32 (2020). https://doi.org/10.1186/s40001-020-00430-5

In November 2020, Borovoy et al. [32] published an extensive review of biological and medical knowledge that allowed them to infer a large potential for significant harms from masking, via microbial challenges from the masks. They rightly stress the known yet underplayed role of bacteria in viral pandemics, and also review respiratory diseases arising from oral bacteria, which can be induced by mask wearing to penetrate and infect the respiratory tract and lungs.

[32] 2020--Borovoy : Boris Borovoy, Colleen Huber, Maria Crisler. “**Masks, false safety and real dangers, Part 2: Microbial challenges from masks**”. *Primary Doctor Medical Journal*. November 2020. - https://pdmj.org/

Endnotes / References

[1] 2020--Hickey and Rancourt : "**21 June 2020 letter to the Executive Director of the WHO. RE: WHO advising the use of masks in the general population to prevent COVID-19 transmission**". Hickey, J and Rancourt DG. *Ontario Civil Liberties Association* (21 June 2020). http://ocla.ca/ocla-letter-who/

[2] 2020--Lazzarino : "Rapid Response: **Covid-19: important potential side effects of wearing face masks that we should bear in mind**". Antonio Lazzarino, A Steptoe, M Hamer, S Michie. 20 April 2020. *BMJ*. ---- https://www.bmj.com/content/369/bmj.m1435/rr-40

[3] 2020--Frountzas : M. Frountzas, C. Nikolaou, D. Schizas et al., "**Personal protective equipment against COVID-19: Vital for surgeons, harmful for patients?**", The American Journal of Surgery. 13 August 2020. https://doi.org/10.1016/j.amjsurg.2020.09.014

[4] 2021--Vainshelboim : Vainshelboim B. "**Facemasks in the COVID-19 era: A health hypothesis**". *Medical Hypotheses*. 2021;146:110411. doi:10.1016/j.mehy.2020.110411 ---- https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7680614/

[5] 2020--Rancourt : "**Masks Don't Work: a Review of Science Relevant to Covid-19 Social Policy**". Rancourt, DG (11 April 2020) *ResearchGate*, obtained 400 K reads, then was deplatformed, as per this report: https://archive.org/details/covid-censorship-at-research-gate-2/ . Now at: https://vixra.org/abs/2006.0044 , and at: https://www.rcreader.com/commentary/masks-dont-work-covid-a-review-of-science-relevant-to-covide-19-social-policy . And see the *Digi-Debates* about criticism of the article: "**Digi-Debates. The Face Mask Debate**", Digi Debates YouTube Channel, 25 July 2020, https://youtu.be/AQyLFdoeUNk , and at: https://www.digi-debates.com/ .

[6] 2020--Rancourt : "**Face masks, lies, damn lies, and public health officials: "A growing body of evidence"**". ResearchGate (3 August 2020). DOI: 10.13140/RG.2.2.25042.58569 - https://www.researchgate.net/publication/343399832_Face_masks_lies_damn_lies_and_public_health_officials_A_growing_body_of_evidence

[7] 2020--Rancourt : "**Measures do not prevent deaths, transmission is not by contact, masks provide no benefit, vaccines are inherently dangerous: Review update of recent science relevant to COVID-19 policy**". Rancourt, DG (28 December 2020). Republished, *PANDA* (3 January 2021). https://www.pandata.org/science-review-denis-rancourt/

[8] 2020--Bakhit : "**Downsides of face masks and possible mitigation strategies: a systematic review and meta-analysis**". Mina Bakhit, Natalia Krzyzaniak, Anna Mae Scott, Justin Clark, Paul Glasziou, Chris Del Mar. *medRxiv* 2020.06.16.20133207; doi: https://doi.org/10.1101/2020.06.16.20133207. Now accepted for publication in *BMJ* Open. ---- https://www.medrxiv.org/content/10.1101/2020.06.16.20133207v1

[9] 2021--Galanis : Galanis P, Vraka I, Fragkou D, Bilali A, Kaitelidou D. "**Impact of personal protective equipment use on health care workers' physical health during the COVID-19 pandemic: a systematic review and meta-analysis**". *medRxiv*; 2021. DOI: 10.1101/2021.02.03.21251056. ---- https://www.medrxiv.org/content/medrxiv/early/2021/02/05/2021.02.03.21251056.full.pdf

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My competence to review science about COVID-19

I am retired and a former tenured Full Professor of Physics, University of Ottawa. Full Professor is the highest academic rank. During my 23-year career as a university professor, I developed new courses and taught over 2000 university students, at all levels, and in three different faculties (Science, Engineering, Arts). I supervised more than 80 junior research terms or degrees at all levels from post-doctoral fellow to graduate students to NSERC undergraduate researchers. I headed an internationally recognized interdisciplinary research laboratory, and attracted significant research funding for two decades.

I have been an invited plenary, keynote, or special session speaker at major scientific conferences some 40 times. I have published over 100 research papers in leading peer-reviewed scientific journals, in the areas of physics, chemistry, geology, bio-geochemistry, measurement science, soil science, and environmental science.

My scientific h-index impact factor is 40, and my articles have been cited more than 5,000 times in peer-reviewed scientific journals (profile at Google Scholar: https://scholar.google.ca/citations?user=1ChsRsQAAAAJ).

My personal knowledge and ability to evaluate the facts in this article are grounded in my education, research, training and experience, as follows:

i. *Regarding environmental nanoparticles.* Viral respiratory diseases are transmitted by the smallest size-fraction of virion-laden aerosol particles, which are reactive environmental nanoparticles. Therefore, the chemical and physical stabilities and transport properties of these aerosol particles are the foundation of the dominant contagion mechanism through air. My extensive work on reactive environmental nanoparticles is internationally recognized, and includes: precipitation and growth, surface reactivity, agglomeration, surface charging, phase transformation, settling and sedimentation, and reactive dissolution. In addition, I have taught the relevant fluid dynamics (air is a compressible fluid), and gravitational settling

at the university level, and I have done industrial-application research on the technology of filtration (face masks are filters).

ii. *Regarding molecular science, molecular dynamics, and surface complexation.* I am an expert in molecular structures, reactions, and dynamics, including molecular complexation to biotic and abiotic surfaces. These processes are the basis of viral attachment, antigen attachment, molecular replication, attachment to mask fibers, particle charging, loss and growth in aerosol particles, and all such phenomena involved in viral transmission and infection, and in protection measures. I taught quantum mechanics at the advanced university level for many years, which is the fundamental theory of atoms, molecules and substances; and in my published research I developed X-ray diffraction theory and methodology for characterizing small material particles.

iii. *Regarding statistical analysis methods.* Statistical analysis of scientific studies, including robust error propagation analysis and robust estimates of bias, sets the limit of what reliably can be inferred from any observational study, including randomized controlled trials in medicine, and including field measurements during epidemics. I am an expert in error analysis and statistical analysis of complex data, at the research level in many areas of science. Statistical analysis methods are the basis of medical research.

iv. *Regarding mathematical modelling.* Much of epidemiology is based on mathematical models of disease transmission and evolution in the population. I have research-level knowledge and experience with predictive and exploratory mathematical models and simulation methods. I have expert knowledge related to parameter uncertainties and parameter dependencies in such models. I have made extensive simulations of epidemiological dynamics, using standard compartmental models (SIR, MSIR) and new models.

v. *Regarding measurement methods.* In science there are five main categories of measurement methods: (1) spectroscopy (including nuclear, electronic and vibrational spectroscopies), (2) imaging (including optical and electron microscopies, and resonance imaging), (3) diffraction (including X-ray and neutron diffractions, used to elaborate molecular, defect and magnetic structures), (4) transport measurements (including reaction rates, energy transfers, and conductivities), and (5) physical property measurements (including specific density, thermal capacities, stress response, material fatigue…). I have taught these measurement methods in an interdisciplinary graduate course that I developed and gave to graduate (M.Sc. and Ph.D.) students of physics, biology, chemistry, geology, and engineering for many years. I have made fundamental discoveries and advances in areas of spectroscopy, diffraction, magnetometry, and microscopy, which have been published in leading scientific journals and presented at international conferences. I know measurement science, the basis of all sciences, at the highest level.

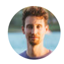
Evaluation of the virulence of SARS-CoV-2 in France, from all-cause mortality 1946-2020

Technical Report · August 2020

DOI: 10.13140/RG.2.2.16836.65920/1



Marine Baudin



Jérémie Mercier

Santé Liberté

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D. G. Rancourt
Ontario Civil Liberties Association

Evaluation of the virulence of SARS-CoV-2 in France, from all-cause mortality 1946-2020

Denis G. Rancourt[1,*], Marine Baudin[2], Jérémie Mercier[2]

[1] Ontario Civil Liberties Association (ocla.ca) ; [2] Mercier Production (jeremie-mercier.com) ;
* denis.rancourt@alumni.utoronto.ca

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20 August 2020

Summary

We analyzed historic and recent all-cause mortality data for France, and other jurisdictions for comparison, using model fitting to quantify winter-burden deaths, and deaths from exceptional events. In this way, COVID-19 is put in historic perspective. We prove that the “COVID-peak” feature that is present in the all-cause mortality data of certain mid-latitude Northern hemisphere jurisdictions, including France, cannot be a natural epidemiological event occurring in the absence of a large non-pathogenic perturbation. We are certain that this “COVID-peak” is artificial because it:

i. occurs sharply (one-month width) at an unprecedented location in the seasonal cycle of all-cause mortality (centered at the end of March),

ii. is absent in many jurisdictions (34 of the USA States have no “COVID-peak”), and

iii. varies widely in magnitude from jurisdiction to jurisdiction in which it occurs.

We suggest that:

- the unprecedented strict mass quarantine and isolation of both sick and healthy elderly people, together and separately, killed many of them,
- that this quarantine and isolation is the cause of the “COVID-peak” event that we have quantified,
- and that the medical mechanism is mainly *via* psychological stress and social isolation of individuals with health vulnerabilities.

According to our calculations, this caused some 30.2 K deaths in France in March and April 2020. However, even including the “COVID-peak”, the 2019-2020 winter-burden all-cause mortality is not statistically larger than usual. Therefore SARS-CoV-2 is not an unusually virulent viral respiratory disease pathogen. By analyzing the all-cause mortality data from 1946 to 2020, we also identified a large and steady increase in all-cause mortality that began in approximately 2008, which is too large to be explained by population growth in the relevant age structure, and which may be related to the economic crash of 2008 and its long-term societal consequences.

Résumé en français

Nous avons analysé les données historiques et récentes de mortalité toutes causes confondues pour la France et d'autres juridictions à des fins de comparaison, en lissant une courbe théorique pour quantifier les décès dus à la charge hivernale et les décès dus à des événements exceptionnels. De cette façon, on peut observer le COVID-19 avec une perspective historique. Ainsi, nous prouvons que le « pic COVID » présent dans les données de mortalité toutes causes confondues de certaines juridictions de l'hémisphère Nord à moyenne latitude, y compris la France, ne peut pas être un événement épidémiologique naturel ayant survenu de façon naturelle, en l'absence d'une grande perturbation non pathogène. Nous sommes convaincus que le « pic COVID » est artificiel car :

i. il s’est produit brusquement (largeur d'un mois) à une date sans précédent dans le cycle saisonnier de mortalité toutes causes confondues (milieu du pic à la fin mars),
ii. il est absent dans de nombreuses juridictions (34 des États américains n'ont pas de « pic COVID »), et
iii. l’ampleur de ce pic varie considérablement d’une juridiction à l’autre.

Nous suggérons que :

- la quarantaine de masse et l'isolement strict sans précédent des personnes âgées malades et en bonne santé, ensemble et séparément, a tué beaucoup d'entre eux,

- que cette quarantaine et cet isolement sont la cause de l'événement « pic-COVID » que nous avons quantifié,
- et que le mécanisme médical expliquant ce pic passe principalement par le stress psychologique et l'isolement social des personnes vulnérables au niveau de leur santé.

Selon nos calculs, ces mesures ont provoqué quelques 30,2 K décès en France en mars et avril 2020. Cependant, même en incluant le « pic COVID », la charge hivernale de mortalité toutes causes confondues pour l’hiver 2019-2020 n'est pas statistiquement supérieure aux charges hivernales habituelles, ce qui nous amène à affirmer que le SARS-CoV-2 n'est pas un virus responsable de maladies respiratoires inhabituellement virulent.

En analysant les données de mortalité toutes causes confondues de 1946 à 2020, nous avons également identifié une augmentation importante et régulière de la mortalité toutes causes confondues qui a commencé vers 2008, trop importante pour être expliquée par la croissance de la population étant donné la pyramide des âges, mais qui pourrait être liée à la crise économique de 2008 et à ses conséquences sociétales sur le long terme.

1. Introduction

France is said to be one of the five European countries most impacted by COVID-19, with Belgium, UK, Italy and Spain.

France has applied broad response measures since the pandemic was declared by the WHO on 11 March 2020, including national lockdown and systematic quarantine of sick and healthy individuals together in care homes and facilities for elderly persons.

The question arises: Is there bias-free hard evidence that the extraordinary measures were and are warranted? After all, if the pathogen is as contagious and virulent as believed, then, irrespective of the array of efforts to mitigate spread of the epidemic, it should be evident by now that the decisions to impose the measures were warranted.

Alternatively, if there is little evidence of an abnormal increase in mortality, then either SARS-CoV-2 is not as dangerous as imagined, or the array of *ad hoc* mitigation measures has been effective and should be considered proven.

2. Data and methods

2.1. Data selection

Cause-of-death assignation and COVID-19 mass “testing” are both susceptible to bias (Cummins, 2020). All-cause mortality is not. Therefore, we use the extensive database of all-cause mortality by month for metropolitan France 1946-2020, and other data (see section 2.2), to cast recent deaths in their historical context. Here, “metropolitan France” means continental France and Corsica (i.e. European France).

2.2. Data retrieval

Table 1 describes the data retrieved and which source it has been collected from.

Data type	Country	Period	Time base	Source
Population	Metropolitan France	1946-2020	Year	Insee (2020c)
All-cause mortality	France	1982-2019	Year	Insee (2020a)
All-cause mortality	Metropolitan France	1946-2020	Month	Insee (2020d)
All-cause mortality	France	1994-2020	Month	Insee (2020e)
All-cause mortality	France	1 March to 20 July for 2018, 2019 and 2020	Day	Insee (2020b)
All-cause mortality	Metropolitan France	1968-2018	Day	Insee (2019)
All-cause mortality	Canada	2014-2020	Week	StatCan (2020)
All-cause mortality	USA	2013-2020	Week	CDC (2020)

Table 1. Data retrieved. Metropolitan France means continental France and Corsica. France means metropolitan France and overseas France.

2.3. Epidemiological data analysis

We chose not to analyse the data by the common method of using a sinusoidal signal intended to separate viral respiratory disease deaths from other seasonally varying

deaths. We believe the latter method, although widely applied, is problematic for the following main reasons:

- The assumed underlying sinusoidal component does not reliably separate deaths assigned as being primarily caused by the viral respiratory disease of interest and the deaths assigned as being primarily due to other seasonally varying (non-viral) causes.
- The sinusoidal model does not correctly fit the non-viral seasonal component of all-cause deaths, since it has systematic residuals in those segments assumed to be unaffected by the viral pathogen.
- There is no biological or medical reason that any seasonal component will have a simple sinusoidal functional form, and many reasons that it would not.

Instead, we analyse the all-cause mortality by month data using a sum of one to three Voigt lines for each peak or feature that rises above the assumed-linear summer baseline for the fitting region. In practice, we select a fitting region over which the summer baseline delimited by the bottoms of the summer troughs is approximately a straight line with a given slope, and use the Voigt lines to fit the peaks that rise above this summer baseline for the fitting region. In this way, the total area of all the Voigt lines in a given winter peak, for example, is the winter-burden mortality for the given winter.

Figure 1 shows that whereas the winter-peak values vary somewhat erratically from year to year, the summer-trough bottoms delineate linear trends with time (the “summer baselines”), in distinct time periods. We delineated the data into five regions as:

2005-2020:	linear with positive slope	“region-I”
1994-2005:	linear with near-zero slope	“region-II”
1968-1994:	linear with near-zero slope	“region-III”
1958-1968:	linear with positive slope	“region-IV”
1946-1958:	linear with near-zero slope	“region-V”

Here, regions II and III both have essentially the same linear summer baselines (**Figure 1**) but were divided into two regions to reduce the sizes of the fittings, and for easier comparison with the 1994-2020 France data (**Figure 2**).

Within each such region (I through V), we fit the data with a linear summer baseline and model peaks for each of the winters. The model peak for a given winter (or a given anomalous peak, see section 3) was taken to be the sum of a variable number, N_{peak}, of Voigt lines. The Voigt lineshape is a convolution between the Lorentzian lineshape and the Gaussian lineshape, such that it can be varied to adopt any shape on a “Lorentzian-Gaussian continuum” of shapes. This is convenient because, for a given lineshape-area, the Lorentzian has broad wings (and a pointed head), whereas the Gaussian shape has a crisp delineation with little wings (and a broad head). The Voigt lineshape is symmetric about its center, whereas all-cause mortality peaks are not generally symmetric, and contain structure such as shoulders, sharp rises, and asymmetric or unequal decays on the two sides. We accommodate such structure by using as many (N_{peak}) Voigt lines in a given all-cause mortality peak as are minimally needed to reduce

the residual (i.e. the difference between the data and the model function) to random noise. With the France 1946-2020 data, this requires between 1 and 3 Voigt lines per peak (N_{peak} = 1 to 3), excluding the anomalous peaks that each require their own Voigt line (one per anomaly, in this case).

Using this method, the winter-burden peaks are well represented and contribute little to raising the summer-trough bottoms above the linear summer baseline. Thus our model reliably captures the winter-burden deaths that occur above the summer baseline. In other words, the winter-burden deaths of a season correspond to the area under the winter-burden peak for that season.

The yearly all-cause mortality is calculated for two types of years: the cycle-year and the calendar-year.

Cycle-year: For a given winter-centered year (cycle-year), the all-cause mortality is equal to the summer baseline value of mortality per month evaluated at the weighted peak position (close to 1 January) times 12 plus the areas of all the N_{peak} Voigt lines in the winter peak.

Calendar-year: The all-cause mortality is obtained by direct counting for the 12 months in each calendar year.

Fitting and quantification are done with the Recoil spectral analysis software, adapted as needed for the epidemiological context (Lagarec and Rancourt, 1998; Rancourt, 2019).

3. Analysis and discussion

3.1. France 1946-2020 data

France maintains a high-quality demographic database, from 1946 to present (Insee, 2020d). **Figure 1** shows all-cause mortality by month for metropolitan France, from January 1946 to June 2020:

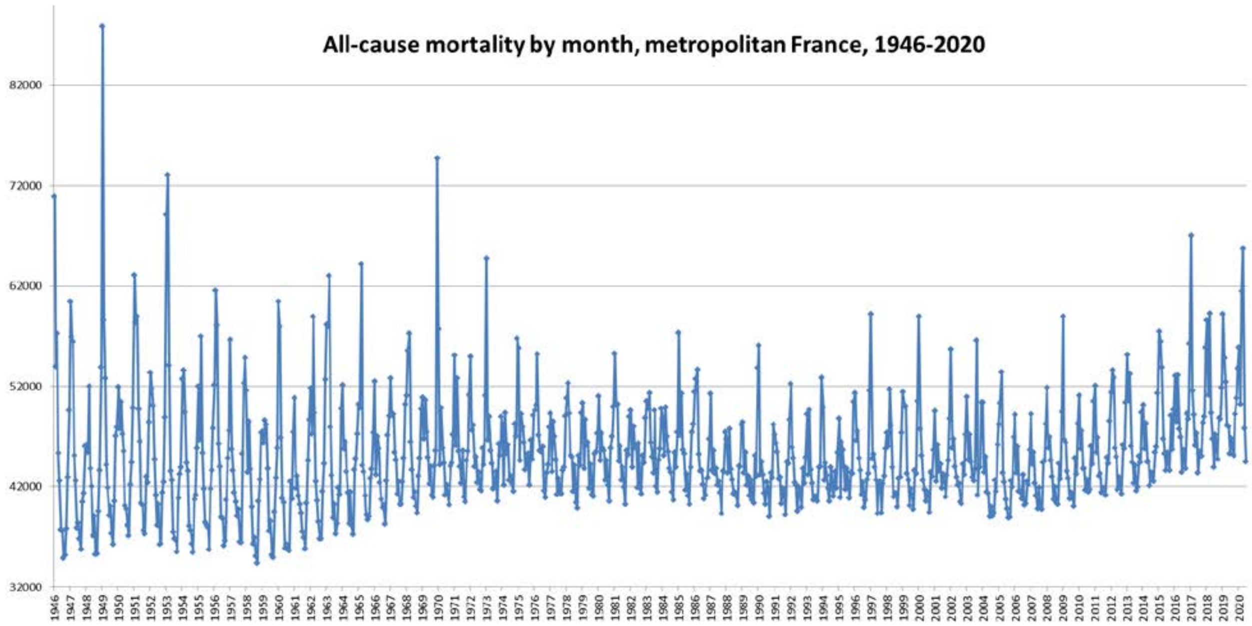


Figure 1. All-cause mortality by month in metropolitan France from 1946 to 2020. Data are displayed from January 1946 to June 2020. Data were retrieved from Insee (**Insee, 2020d)**, as described in **Table 1**.

The data shows the well-known and prominent winter peaks and summer troughs (Dowell, 2001; Marti-Soler *et al.*, 2014; Paules and Subbarao, 2017; Rancourt, 2020). Such seasonal patterns of all-cause mortality occur in all mid-latitude countries. The patterns are shifted by 6 months in the Southern-hemisphere mid-latitudes, where the peaks again correspond to winters in that hemisphere.

Visual inspection of **Figure 1** shows that the 2019-2020 winter mortality in France was not obviously anomalous, at first sight. This is not surprising to us: most provinces in Canada and most states in the USA have 2019-2020 winter-burden all-cause mortalities that are smaller than for each of at least two other winters in the last decade (unpublished).

Figure 1 is a sobering result, which is in contrast to the focus of media coverage since March 2020. There was not an extraordinary winter mortality in France in 2019-2020. In light of 75 years of all-cause mortality data, death has continued its seasonal variation without any remarkable event, remaining within the bounds of year-to-year statistical variation, at least on the large scale of this figure.

In France, there have been five seasons over the last 75 years with a higher maximum in all-cause mortality by month than the maximum of the 2019-2020 season: 1945-1946, 1948-1949, 1952-1953, 1969-1970 and 2016-2017 (**Figure 1**). The 2019-2020 seasonal epidemic was not the worst in a century, as claimed by French president Emmanuel Macron (see France 24, 2020, at 00:34).

3.2. France 1994-2020 data

France has also released "all-France" mortality data, which includes metropolitan and overseas France, for the last nearly three decades (Insee, 2020e). **Figure 2** shows all-cause mortality by month for the whole of France, from January 1994 to June 2020:

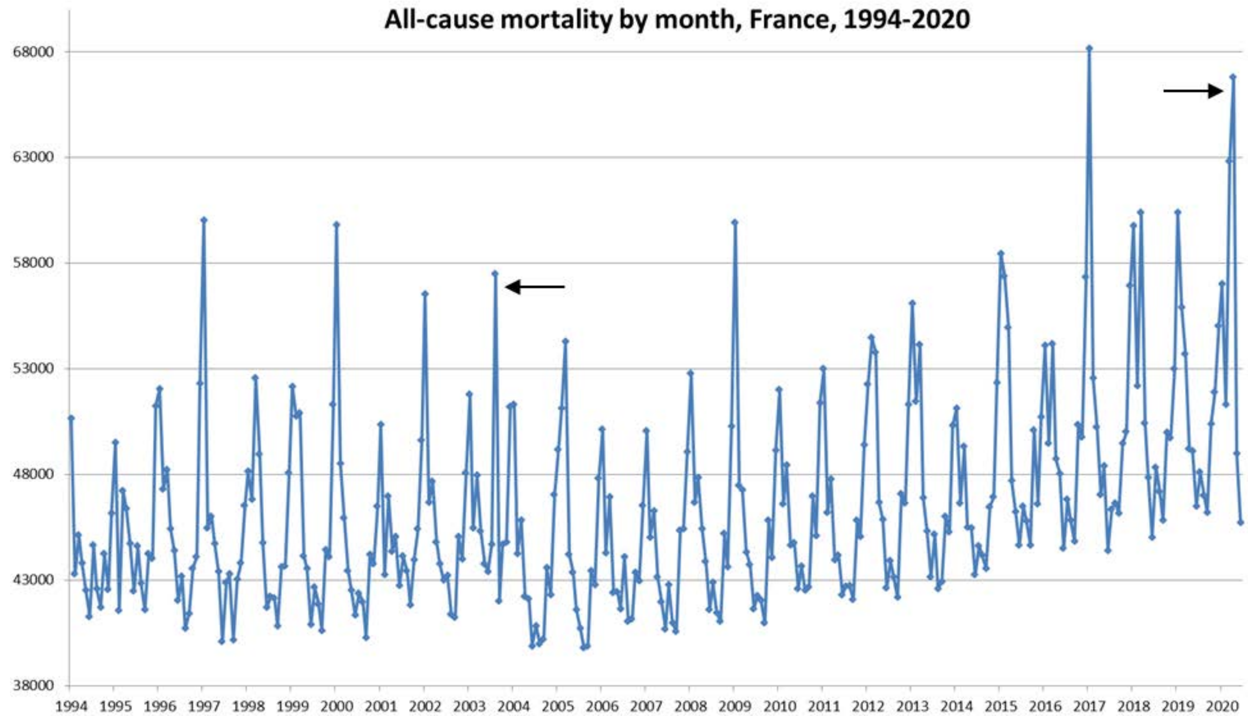


Figure 2. All-cause mortality by month in France from 1994 to 2020. Data are displayed for "all-France", which includes metropolitan and overseas France, from January 1994 to June 2020. The arrows show the two anomalous peaks discussed in the text. Data were retrieved from Insee **(Insee, 2020e)**, as described in **Table 1**.

At this resolution (1994-2020, by month), two anomalies are recognized, which do not conform to known seasonal-variation patterns for mid-latitude countries in the Northern hemisphere: the August-2003 heat wave anomaly and the March-April-2020 anomaly, which we name the "COVID-peak" (following Rancourt (2020)) and describe in the next sections.

3.3. France August-2003 heat wave anomaly

The first anomaly is a single-month spike that occurred in August 2003 ("2003-08"), which would normally be part of a trough in all-cause mortality by month, which rises

near the 58 K deaths/month mark in 2003 (**Figure 2**). This anomaly has conclusively been attributed to an exceptional heat wave that hit nearly all of France in that month and that killed approximately 15 K people (Evin *et al.*, 2004; Hémon and Jougla, 2004). It is an example of deaths that cannot be attributed to a pathogen acting on a population in normal circumstances.

3.4. "COVID-peak" anomaly

The second anomaly is a narrow peak, having a width of approximately 1 month, occurring at (centered on) the end of March 2020, which would normally be the decaying shoulder of the recent winter peak. Winter peaks are always centered at the beginning of January and by March are always in decay towards the next summer trough in all-cause mortality. Rancourt has called the second anomaly the "COVID-peak" and he has postulated that it was caused by the government responses that followed the 11 March 2020 WHO declaration of the pandemic (Rancourt, 2020).

The all-cause mortality by day (**Figure 3**) shows that the said "COVID-peak" occurs on the March-side decay of the preceding winter peaks. **Figure 3** shows the all-cause mortality by day for France, for the years 2018, 2019 and 2020, from 1 March through 30 June:

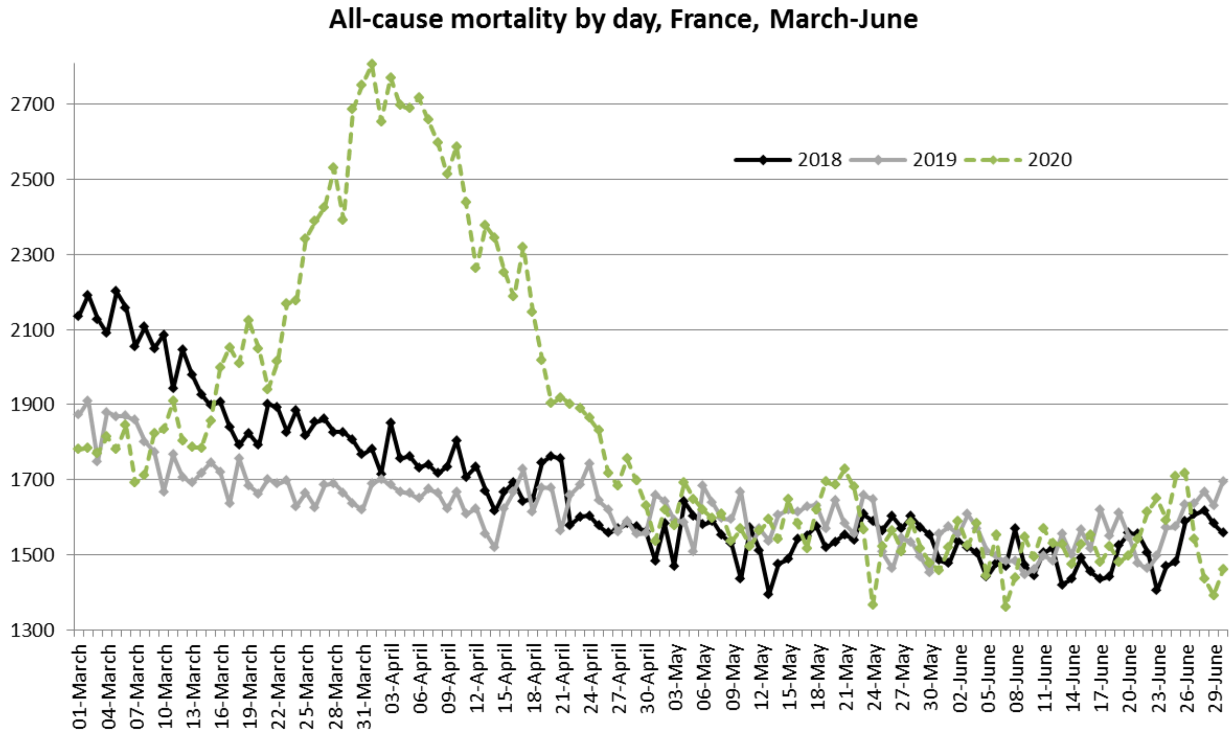


Figure 3. All-cause mortality by day in France from March to June 2018, 2019 and 2020. Data are displayed for "all-France", which includes metropolitan and overseas France, from 1 March to 30 June of 2018, 2019 and 2020. The black line is the data for 2018. The grey line is the data for 2019. The green dashed line is the data for 2020. Data were retrieved from Insee **(Insee, 2020b)**, as described in **Table 1**.

There has never previously been a sharp (1 month width) prominent peak in all-cause mortality, occurring at the end of March, such as this "COVID-peak", in the 75 years of all-cause mortality records for France, nor for available records for Canada and its provinces, the USA and its states, England and Wales, and European countries (Rancourt, 2020 and to be published).

In addition, the "COVID-peak" anomaly not only occurs at a unique time in the epidemiological cycle but also varies widely in magnitude, from zero (e.g. California) to overwhelmingly large (e.g. New York State), in going from one mid-latitude Northern-

hemisphere jurisdiction to another (manuscript in preparation). This is illustrated for Canada, as follows. **Figure 4** shows all-cause mortality by week (number of deaths per week vs standard CDC weeks) from week-1 (first week of January) of 2014 to week-22 (last week of May) of 2020, for the provinces of Ontario and Quebec:

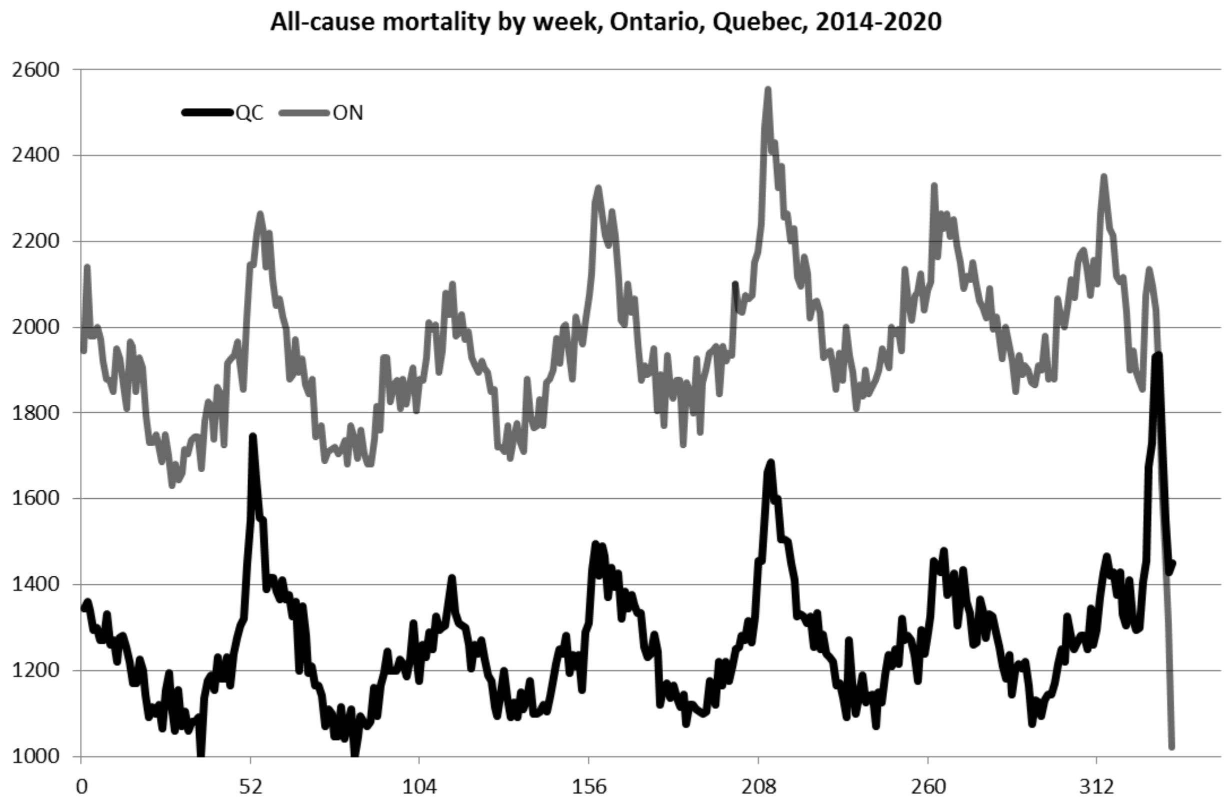


Figure 4. All-cause mortality by week in Ontario and Quebec, from 2014 week-1 to 2020 week-22. The grey line shows the data for Ontario. The black line shows the data for Quebec. Data were retrieved from Statistics Canada **(StatCan, 2020)**, as described in **Table 1**.

Ontario and Quebec are similarly populous East-West adjacent provinces of similar sizes, having distinct medical systems (health is a provincial jurisdiction in the Canadian constitution). As with virtually all mid-latitude Northern-hemisphere countries, the epidemiological cycles (all-cause mortality curves) of Ontario and Quebec are virtually

identical, except for the “COVID-peak” anomaly. The “COVID-peak” is much larger in Quebec than in Ontario, where Quebec was the first province to impose an aggressive lockdown and close its provincial borders.

For decades the epidemiological cycles (all-cause mortality curves) in all mid-latitude Northern-hemisphere jurisdictions have been virtually identical, and have never displayed any peak centered at the end of March, until after 11 March 2020 when a “COVID-peak” anomaly occurred in certain jurisdictions, which is widely variable in magnitude. Therefore, the “COVID-peak” cannot be due to a natural progression of a viral respiratory disease (regardless its virulence), in unperturbed societal structures. Indeed, if this anomaly was due to virulence, it would be difficult to understand the large time-lag between the first reported case in France (27 December 2019 according to Deslandes *et al.*, 2020) and the anomaly’s sudden rise starting in mid-March of the “COVID-peak”. We postulate that the excess all-cause mortality captured by the “COVID-peak” anomaly was caused by government responses to the declaration of the “pandemic” by the WHO on 11 March 2020. It is not a natural epidemiological event, irrespective of the underlying pathogenic and co-morbidity circumstances.

Indeed, the said “COVID-peak” is remarkable in epidemiological terms in that it is entirely absent for many jurisdictions, where the absence appears to be tied more to jurisdictional politics and policy rather than any epidemiological logic. For example, the “COVID-peak” is entirely absent in 34 of the USA States, and varies dramatically in intensity from state to state for those States in which it is present (manuscript in

preparation). **Figure 5** shows a colour-coded map of the USA for “COVID-peak” intensity. Darker green is increased degree of absence of the “COVID-peak”, and darker grey is increased intensity of a discerned “COVID-peak”:

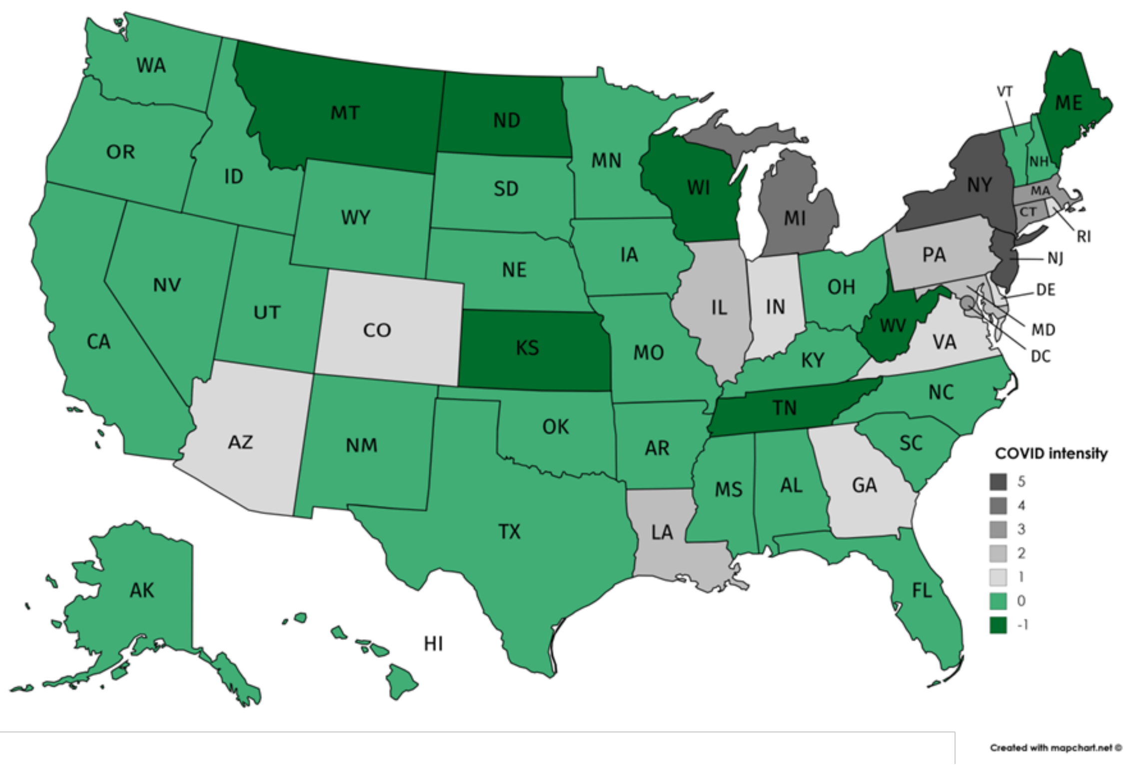


Figure 5. “COVID-peak” intensity map of the USA. States in green are the states where the “COVID-peak” is absent. The darker the green, the more intense the absence. States in grey are the states where the “COVID-peak” is present. The darker the grey, the more intense the presence. Data were retrieved from CDC **(CDC, 2020)**, as stated in **Table 1**.

Here, all the USA States have comparable infection rates, according to reported mass testing results (Ioannidis, 2020). Such geographical variation in an all-cause mortality peak that occurs simultaneously in various localities on two continents is unprecedented in the natural history of human epidemiology.

Either SARS-CoV-2 is such a unique viral respiratory disease pathogen, unlike any previously seen, that it can naturally cause a mortality peak at the end of March, across the mid-latitude Northern-hemisphere world, solely in certain jurisdictions where it occurs, or synchronous and local external (non-pathogenic) factors played a major role. We conclude the latter.

3.5. Quantitative analysis of the all-cause mortality data

Next, we made a quantitative analysis of the all-cause mortality by month for metropolitan France from January 1946 to June 2020 (**Figure 1**), as described in section 2.3.

Figure 6 shows our fit, and its residual, for region-II (1994-January through 2005-September):

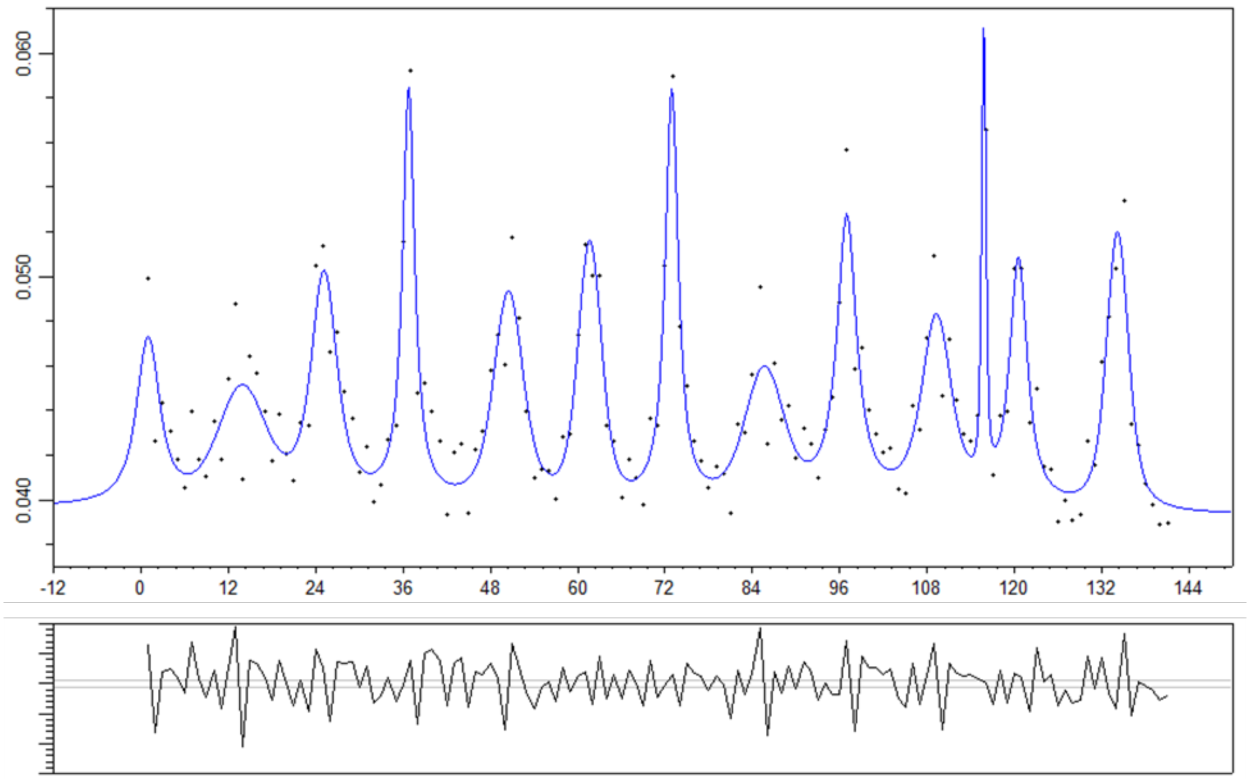


Figure 6. Fit of the monthly all-cause mortality data of metropolitan France 1946-2020, region-II. Region-II corresponds to the period between January 1994 and September 2005, as defined in section 2.3. The y-scale is millions of deaths per month. The x-scale is in months. The blue line is the fitted function. The residual is shown at the bottom.

The single-month spike that corresponds to the August-2003 heat wave is seen at month number 116, and, in our fit, corresponds to a spike area (heat wave deaths) of 19 K deaths. Note that our goal here was not to determine an accurate number of deaths for the heat wave itself but rather to correctly represent the total mortality profile in this period. We obtain a more accurate value of 15.3 K deaths for this heat wave by our analysis of the higher resolution all-cause mortality by day (Insee, 2019) (not shown). The difference (19 K versus 15.3 K) occurs because higher resolution data provides greater power to separate overlapping contributions in a given region of the data.

Figure 7 shows our fit, and its residual, for region-I (2005-August through 2020-June):

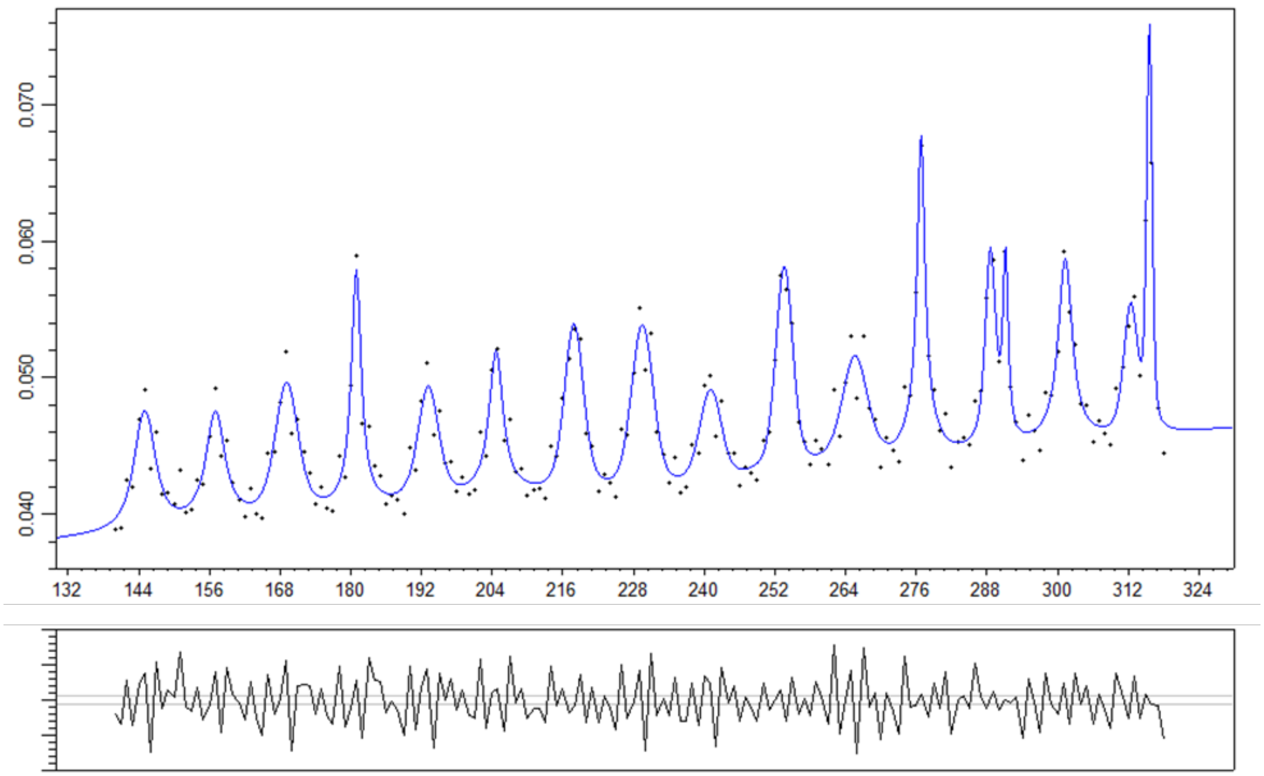


Figure 7. Fit of the monthly all-cause mortality data of metropolitan France 1946-2020, region-I. Region-I corresponds to the period between August 2005 and June 2020, as defined in section 2.3. The y-scale is millions of deaths per month. The x-scale is in months. The blue line is the fitted function. The residual is shown at the bottom.

The month-wide "COVID-peak" is seen, centered at the end of March 2020, straddling March and April, as seen in **Figure 3**. In this fit (**Figure 7**), the "COVID-peak" has an estimated area of 41 K deaths. This estimate is limited in accuracy by two main factors: (i) the low temporal resolution of the mortality by month data, which limits the power to separate overlapping contributions, and (ii) the missing mortality by month data beyond June 2020. These problems are resolved in our analysis of the mortality by day data, as follows.

Accurate quantification of the deaths in the complete “COVID-peak” is obtained by fitting the all-cause mortality by day for France for 1 March 2020 through 30 June 2020, shown in **Figure 3**. The fit uses a linear sloped background for the non-COVID-peak components and two Voigt lines (N_{peak} = 2) for the “COVID-peak”, as shown in **Figure 8**:

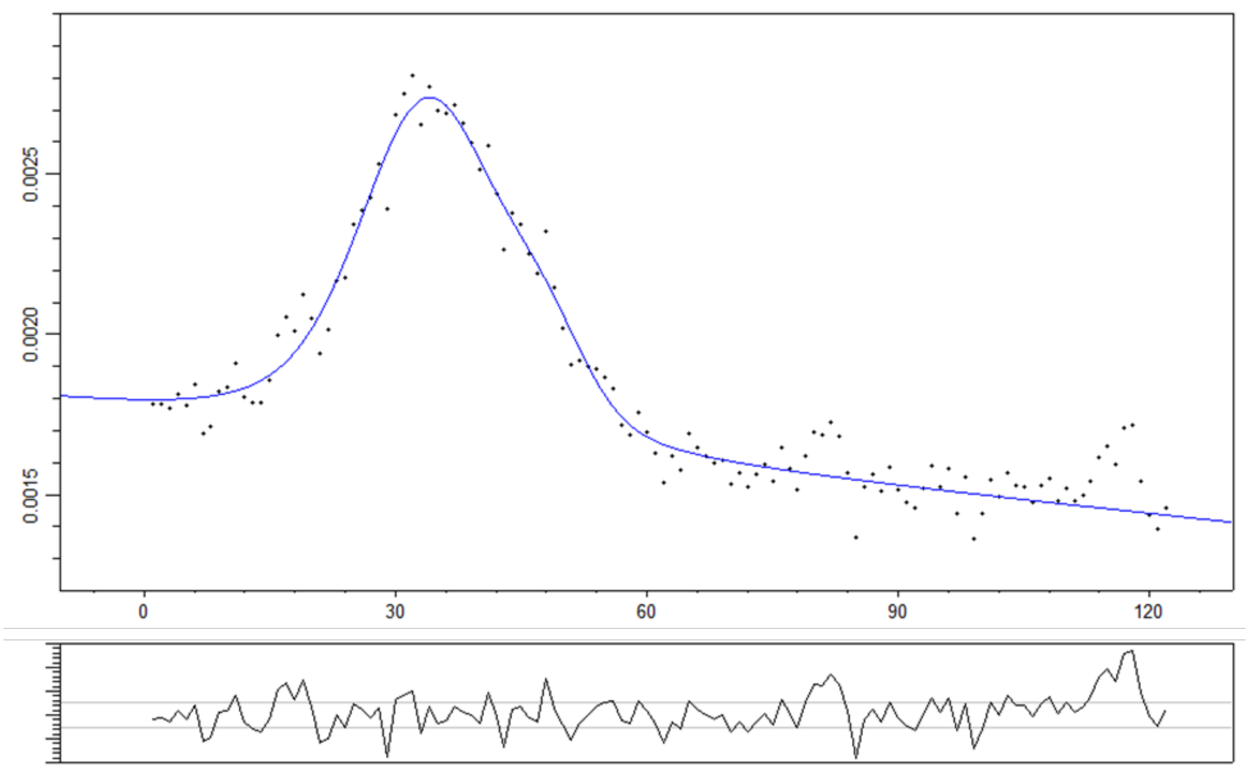


Figure 8. Fit of the daily all-cause mortality data of France (metropolitan + overseas), from 1 March to 30 June 2020. The y-scale is millions of deaths per day. The x-scale is in days. The blue line is the fitted function. The residual is shown at the bottom.

This fit gives an accurate “COVID-peak” area equal to 30.2 K deaths, which is approximately double the deaths from the August-2003 heat wave in France, and which we attribute to the total deaths in France due to government interventions responding to the declared “pandemic”.

3.6. Graphical analysis of the model-fitting results

In examining our fit results for metropolitan France 1946-2020, we first calculate the all-cause mortality per cycle-year, as defined in section 2.3.

Figure 9 shows the all-cause mortality per cycle-year for metropolitan France 1946-2020, compared to the all-cause mortality per calendar-year for the same data:

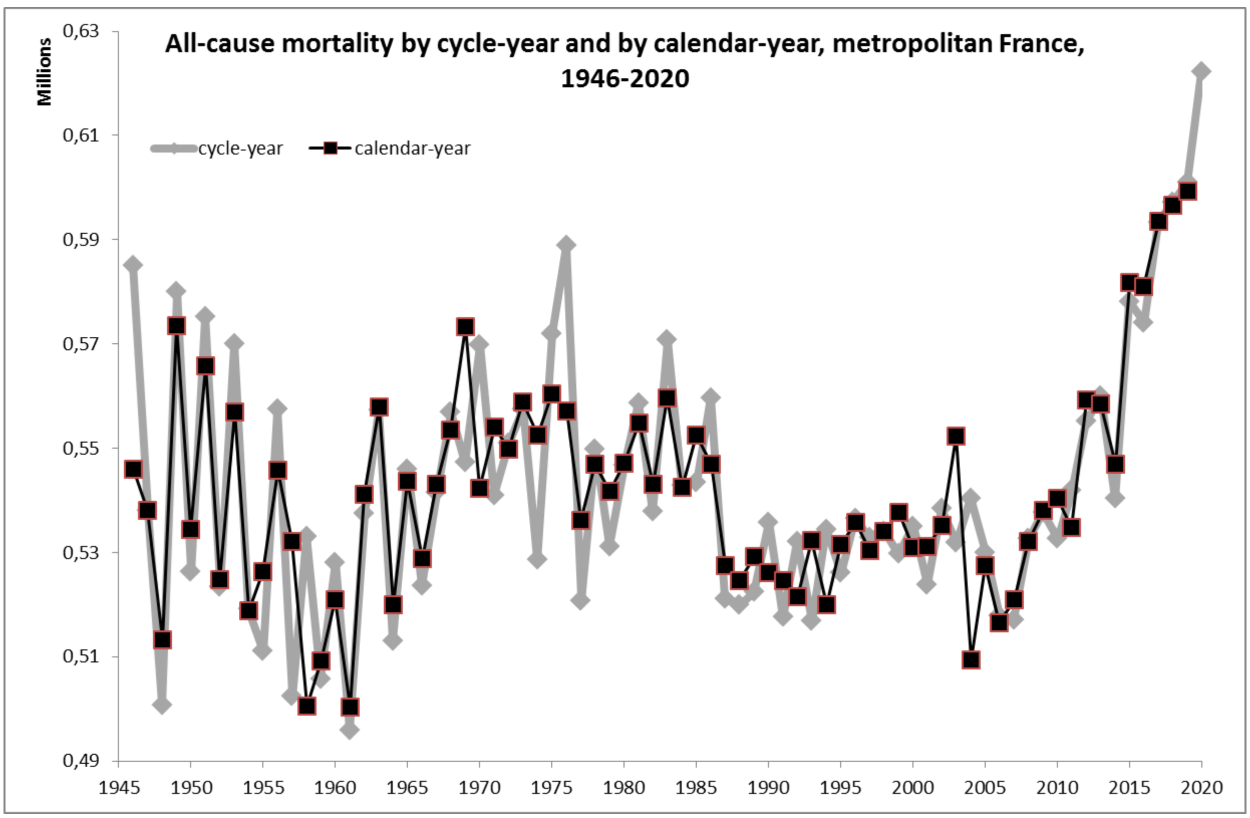


Figure 9. All-cause mortality by cycle-year and by calendar-year in metropolitan France from 1946 to 2020. The grey line shows the data per cycle-year (centered in January), meaning that the year of the month of January in the winter peak is used on the x-axis. The black line shows the data per calendar-year (direct sum). The cycle-year values were obtained by fitting, as described in section 2.3.

The break that occurs between 1986 and 1987 is probably an artifact of the data collection method. There may be another such break between 1961 and 1962. Overall, there is a decline of mortality per year after the Second World War and up to 1961, plateaus in mortality per year for the periods 1962-1986 and 1987-2008, and a steady and steep increase starting at approximately 2008 through to the present. The latter steady and steep increase is essentially the same as reported by Insee (2020a) for the yearly mortality data for France, 1982-2019.

The latter 2008-present rise in all-cause mortality per year is remarkable, approximately double than can be accounted for by the increasing population with a constant age structure. How is this dramatic break and increase, which also occurs in Canada and the USA, not a “pandemic”? It has not attracted any media attention, to our knowledge. Was it caused by the global economic crash of 2008, which many economists compare to the Great Depression (Bordo and James, 2009; Shaikh, 2010; Chang *et al.*, 2013; O’Brien, 2018)? There is a surprising media and academic-research relative silence regarding this compelling public health phenomenon (**Figure 9**), although some research for other countries is tangentially relevant (e.g., Falagas *et al.*, 2009; Stuckler *et al.*, 2009; Ruhm, 2016).

Figure 10 shows the all-cause mortality in metropolitan France per cycle-year (as defined in section 2.3), as a percentage of the population of metropolitan France evaluated on 1 January of each year, for the 1946-2020 period:

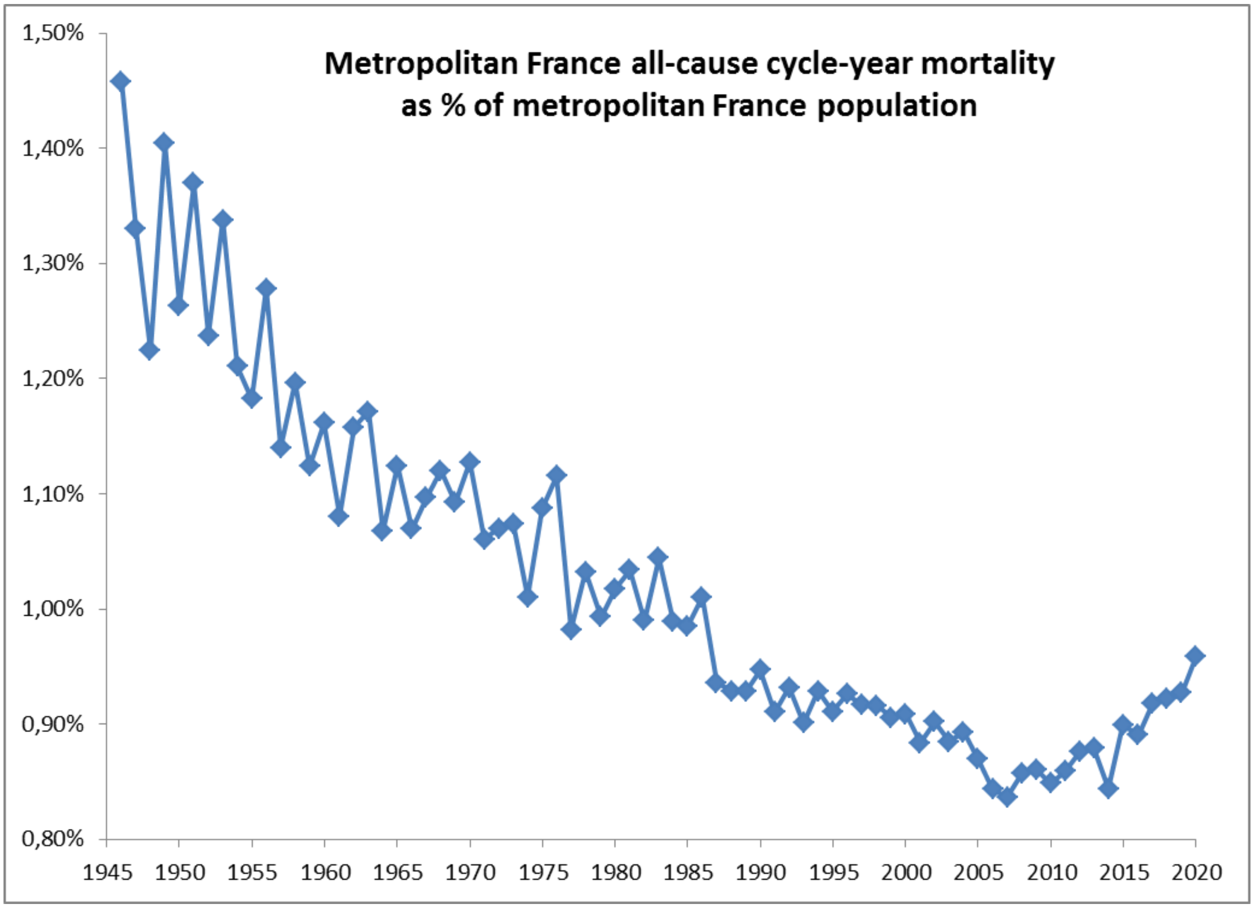


Figure 10. All-cause mortality by cycle-year in metropolitan France from 1946 to 2020, as a percentage of French metropolitan population over the same period. The x-axis year is the year of the January in the cycle-year (the January of the winter season). The population is for 1 January of each year. The population data was retrieved from Insee **(Insee, 2020c)**, as stated in **Table 1**.

Again, we note the dramatic upturn at approximately 2008. Mortality on a per capita basis decreases steadily after the Second World War, and then the trend is reversed to increasing mortality, starting at approximately 2008.

The estimate of cycle-year mortality for nominally 2020 is expected to be fairly good because the fit (**Figure 7**) reasonably completes the 2019-2020 winter peak, down to the expected 2020 summer trough (and see **Figure 3**).

With **Figure 10**, it is difficult to see the latest winter cycle that includes the “COVID-peak” as extraordinary. The value does not appear to warrant any extreme reaction, in the context of the entire 1946-2020 trend and its both regular and statistical variations.

By comparison, the upturn in yearly all-cause mortality, which is initiated at approximately 2008, is real and does warrant public concern and a public-health investigation. It seems unreasonable to concentrate on an external-event disaster (“COVID-peak”), while ignoring a massive and systematic health issue easily detected after analyzing all-cause mortality data.

Figure 11 shows the numbers of winter-burden deaths for metropolitan France 1946-2020, which result from our fits of the data for all-cause mortality by month:

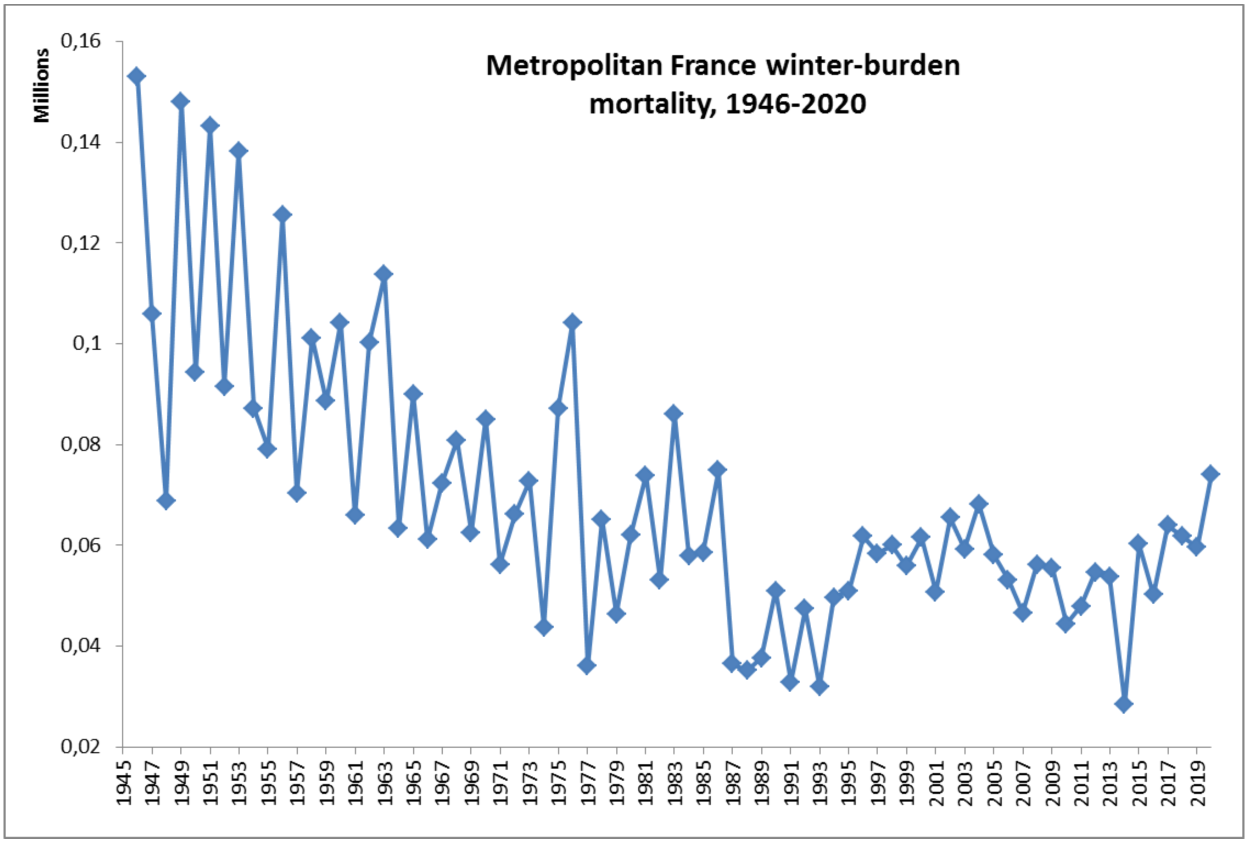


Figure 11. Winter-burden mortality in metropolitan France from 1946 to 2020. The data results from the fit of monthly all-cause mortality in metropolitan France, 1946-2020. The x-axis year is the year of the January in the cycle-year (the January of the winter season).

In **Figure 12**, the same numbers of winter-burden deaths for metropolitan France 1946-2020, which result from our fits of the data for all-cause mortality by month, are expressed as percentages of the total all-cause mortality per cycle-year, for each given cycle-year having its own winter-burden mortality:

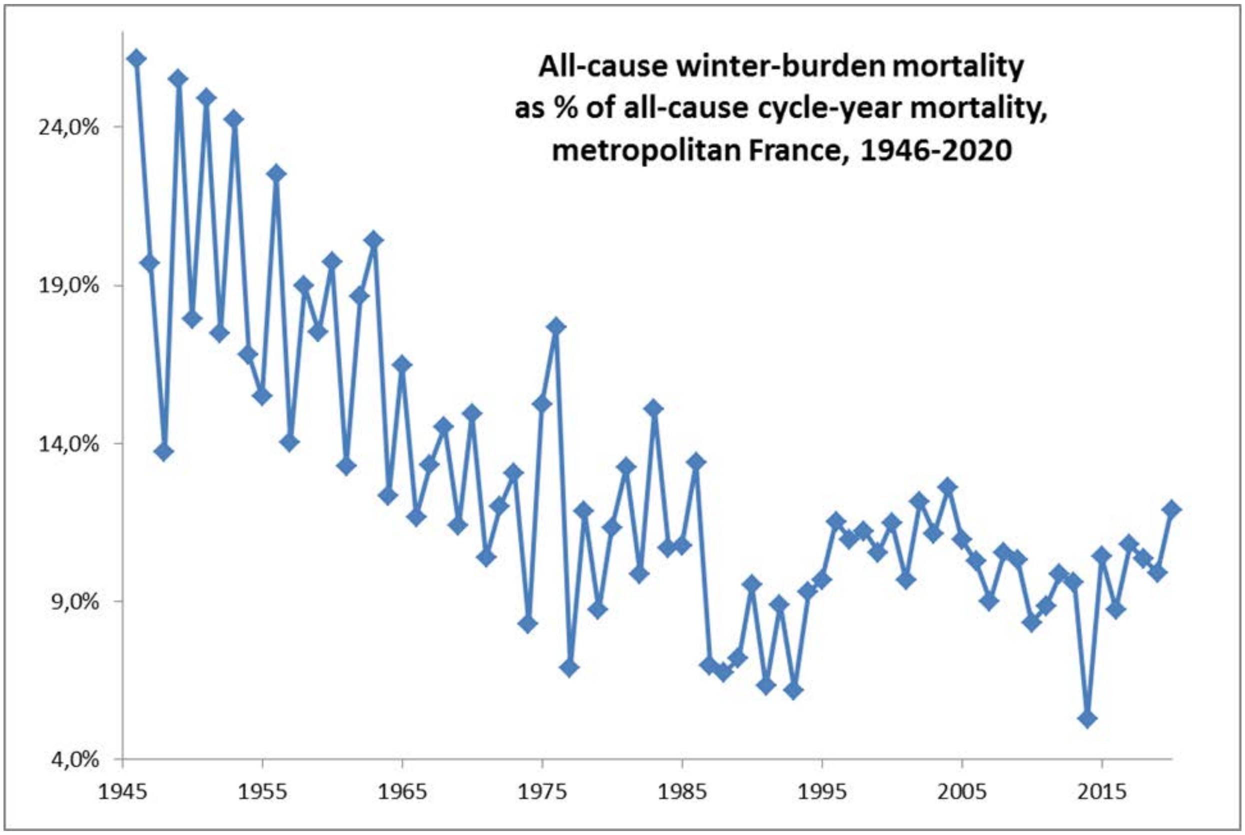


Figure 12. All-cause winter-burden mortality as a percentage of yearly all-cause mortality in metropolitan France from 1946 to 2020. The data are cycle-year based (see section 2.3). The x-axis year is the year of the January in the cycle-year (the January of the winter season).

The anti-correlation in time for year to year values (a low year is followed by a high year, and a high year is followed by a low year), especially prominent in the early years following the Second World War, seen in both **Figure 11** and **Figure 12**, is real and can be interpreted as follows: winter-burden mortality is a convolution between the prevailing pathogenic conditions and the population of immune-vulnerable individuals (i.e. population of fragile mostly elderly persons). A winter that relatively devastates the fragile-person population leaves a relatively small such population for the following winter, and *vice versa*. The year-to-year effect is greatest to the extent that the mean lifetime of a concerned fragile person is one year. In other words, the one-year time

anti-correlation is predominantly from the number of individuals having a one-year mean lifetime or life expectancy.

This shows that it would be ill-advised to assign such year-to-year variations in winter-burden mortality to virulence of the particular year's seasonal viral pathogens. The changes are more a function of the general health status of the population, and the population numbers of the most vulnerable individuals, rather than virulence of a particular pathogen. It would be incorrect to postulate that viral virulence progressively decreased after the Second World War in France, just as it would be incorrect to interpret relatively small variations occurring in recent decades as being due to year-to-year changes in virulence of the seasonal pathogens.

Figure 12 shows that 2019-2020 was not a statistically unusual cycle-year in France, in terms purely of the total number of winter-burden deaths, which include the anomalous "COVID-peak" deaths. Is this because mitigation measures were effective in the presence of an exceptionally virulent pathogen? On the contrary, as explained above, the "COVID-peak" anomaly must be interpreted as the result of an exceptional imposed perturbation in the society. The "COVID-peak" would not have occurred in the absence of the said perturbation, and some 30.2 K lives would have been saved in France.

4. Mechanistic causes for “COVID-peak” deaths

In light of epidemiological history, we have proven that the “COVID-peak” feature that is present in the all-cause mortality data of certain mid-latitude Northern hemisphere jurisdictions, including France, cannot be a natural epidemiological event occurring in an absence of an external non-pathogenic perturbation. This is true because the “COVID-peak”:

i. occurs sharply (one-month width) at an unprecedented location in the seasonal cycle (centered at the end of March),
ii. is absent in many jurisdictions (34 of the USA States have no “COVID-peak”), and
iii. varies widely in magnitude from jurisdiction to jurisdiction in which it occurs (such as the example of Ontario and Quebec, **Figure 4**).

Such a feature in all-cause mortality by week or month has never previously occurred in known epidemiological data, except with exceptional events such as the August-2003 heat wave in France, or regional earthquakes. Barring such exceptional events, the known all-cause mortality curves for populations in the entire mid-latitude Northern hemisphere are remarkably the same; without disappearing or appearing peaks in different geographical locations, and without peaks occurring at unusual times in the seasonal cycles.

We end this article by outlining a mechanism wherein one aspect of government responses could have caused the excess 30.2 K deaths in the “COVID-peak”.

We believe that the unprecedented strict mass quarantine and isolation of both sick and healthy elderly people, together and separately, would have killed many of them, and is the main cause of the “COVID-peak” event that we have identified.

By the said mass quarantine in care homes and establishments, the State isolated vulnerable elderly persons from their families, limited movements within establishments, often confining individuals to their rooms or beds for days and weeks if not months, reduced the staff and allowed staff to take extended or frequent sick leaves, forced staff to adopt extreme measures such as masks, shields and gloves, which can induce a measure of fear or terror, created a general atmosphere of danger, and prevented air circulation by locking doors and windows, and by preventing ingoing and outgoing traffic except for essential services (Campbell, 2020; Comas-Herrera, Fernandez, *et al.*, 2020; Wu, 2020).

This would have both: retained the pathogen-bearing aerosol particles suspended in the air without their evacuation (Morawska and Milton, 2020); and induced psychological stress in the residents.

Psychological stress is known:

i. to be a major factor causing diseases, including immune response dysfunction, depression, cardiovascular disease and cancer (Cohen, Janicki-Deverts and Miller, 2007),

ii. to be a dominant factor in making an individual susceptible to viral respiratory diseases, in terms of intensity of the infection (Cohen, Tyrrell and Smith, 1991), and

iii. to have more deleterious effects in elderly persons than in younger persons (Prenderville *et al.*, 2015).

Furthermore, social isolation itself, in addition to individual psychological stress, is known to have an added impact on the said susceptibility to viral respiratory disease (Cohen *et al.*, 1997).

In addition, there is a longer term “abandonment of life” phenomenon that occurs with imposed extended isolations of elderly persons, the so-called “glissement” syndrome (or “slipping away syndrome” or “geriatric failure to thrive”), which is analogous to depression (Robertson and Montagnini, 2004; Clegg *et al.*, 2013; Steptoe *et al.*, 2013; Ong, Uchino and Wethington, 2016).

The suddenly applied national policy of forced quarantine and the psychological stress it generated on fragile elderly people was certainly a major contributor in the decrease of efficiency of immune system response to a viral respiratory disease (Comas-Herrera,

Zalakaín, *et al.*, 2020) and this is today the most probable explanation for the most part of the sharp and narrow mass excess death peak that occurred in March-April 2020 in France. The same mechanism would operate in any setting (facility, group home, home, hospital) where persons with health vulnerabilities are isolated and susceptible to psychological stress.

We claim that this mechanism is what occurred, as first suggested by Rancourt (2020), and that this caused some 30.2 K deaths in France in March and April 2020, not any viral respiratory disease or combination of such acting naturally in an unperturbed society.

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Face masks, lies, damn lies, and public health officials: "A growing body of evidence"

Denis G. Rancourt, PhD
Researcher, Ontario Civil Liberties Association (ocla.ca)

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Summary

A vile new mantra is on the lips of every public health official and politician in the global campaign to force universal masking on the general public: "there is a growing body of evidence".

This propagandistic phrase is a vector designed to achieve five main goals:

- Give the false impression that a balance of evidence now proves that masks reduce the transmission of COVID-19
- Falsely assimilate commentary made in scientific venues with "evidence"

- Hide the fact that a decade's worth of policy-grade evidence proves the opposite: that masks are ineffective with viral respiratory diseases
- Hide the fact that there is now direct observational proof that cloth masks do not prevent exhalation of clouds of suspended aerosol particles; above, below and through the masks
- Deter attention away from the considerable known harms and risks due to face masks, applied to entire populations

The said harms and risks include that a cloth mask becomes a culture medium for a large variety of bacterial pathogens, and a collector of viral pathogens; given the hot and humid environment and the constant source, where home fabrics are hydrophilic whereas medical masks are hydrophobic.

In short, I argue: op-eds are not "evidence", irrelevance does not help, and more bias does not remove bias. Their mantra of "a growing body of evidence" is a self-serving contrivance that impedes good science and threatens public safety.

I prove that there is no policy-grade evidence to support forced masking on the general population, and that all the latest-decade's policy-grade evidence points to the opposite: NOT recommending forced masking of the general population. Therefore, the politicians and health authorities are acting without legitimacy and recklessly.

The article is organized into the following sections:

Introduction

On 5 June 2020, the World Health Organization (WHO) reversed more than a decade of public health bodies around the world expressly not recommending face masks for the general population. [1]

The WHO made its recommendation of the preventative medical intervention of face masks for the entire global population while stating: [2]

> “At the present time, the widespread use of masks by healthy people in the community setting is not yet supported by high quality or direct scientific evidence and there are potential benefits and harms to consider (see below).” (p. 6)

The pretext used by the WHO was:

> “a growing compendium of observational evidence on the use of masks by the general public in several countries”. (p. 6)

Therefore, in its recommendation that could have devastating civil, social and medical consequences, when enforced on the scale of the world population, the WHO violated the Golden Rule of medical ethics: "You don't recommend an intervention without policy-grade evidence for both harms and benefits".

Regarding the said Golden Rule of medical ethics, allow me to quote the most authoritative voices of Califf, Hernandez and Landray, discussing medical-treatment-protocol assessment during COVID-19, and writing in the prestigious *Journal of the American Medical Association* (*JAMA*) on 31 July 2020: [3]

> […] However, there is growing concern about whether attempts to infer causation about the benefits and risks of potential therapeutics from nonrandomized studies are providing insights that improve clinical knowledge and accelerate the search for needed answers, or whether these reports just add noise, confusion, and false confidence. <u>Most of these studies include a caveat indicating that "randomized clinical trials are needed." But disclaimers aside, does this approach help make the case for well-designed randomized clinical trials (RCTs) and accelerate their delivery? Or do observational studies reduce the likelihood of a properly designed trial being performed, thereby delaying the discovery of reliable truth?</u>
> […]
> Anxious, frightened patients, as well as clinicians and health systems with a strong desire to prevent morbidity and mortality, are all susceptible to cognitive biases. Furthermore, profit motives in the medical products industry, academic hubris, interests related to increasing the valuation of data platforms, and revenue generated by billing for these products in care delivery can all tempt investigators to make claims their methods cannot fully support, and these claims often are taken up by traditional media and further amplified on social media. Politicians have been directly involved in discourse about treatments they assert are effective. <u>The natural desire of all elements of society to find effective therapies can obscure the difference between a proven fact and an exaggerated guess</u>. Nefarious motives are not necessary for these problems to occur.

> […] But if leaders, commentators, academics, and clinicians cannot restrain the rush to judgment in the absence of reliable evidence, the proliferation of observational treatment comparisons will hinder the goal of finding effective treatments for COVID-19—and a great many other diseases.

Thus, we see that the WHO and local public health officials are hindering advancement, by promoting non-RCT “observational studies”, rather than protecting public health.

It should be of great concern to all that the WHO pretext of “a growing compendium of observational evidence on the use of masks by the general public in several countries” has morphed into the mantra “a growing body of evidence”, which finds itself on the lips of virtually all public health officers and city mayors in the country.

This mantra of “a growing body of evidence” is advanced as the false silver bullet justification for draconian masking laws, in actual circumstances in which:

- There have been NO new RCT studies that support masking
- All the many past RCT studies conclusively do not support masking
- None of the known harms of masking have been studied
  (re: enforcement on the entire general population)

This is the opposite of science-based policy. The politicians and public health officers are actuating the worst decisional model that can be applied in a rational and democratic society: forced preventative measures without a scientific basis, while recklessly ignoring consequences.

In this article, I prove that there is no policy-grade evidence to support forced masking on the general population, and that all the latest decade's policy-grade evidence points to the opposite: NOT recommending forced masking of the general population.

Therefore, the politicians and health authorities are acting without legitimacy and recklessly.

Competence to talk about face masks and COVID-19

I am retired and a former tenured Full Professor of Physics, University of Ottawa. Full Professor is the highest academic rank. During my 23-year career as a university professor, I developed new courses and taught over 2000 university students, at all levels, and in three different faculties (Science, Engineering, Arts). I supervised more than 80 junior research terms or degrees at all levels from post-doctoral fellow to graduate students to NSERC undergraduate researchers. I headed an internationally recognized interdisciplinary research laboratory, and attracted significant research funding for two decades.

I have been an invited plenary, keynote, or special session speaker at major scientific conferences some 40 times. I have published over 100 research papers in leading peer-reviewed scientific journals, in the areas of physics, chemistry, geology, materials

science, soil science, and environmental science. I have made fundamental scientific discoveries in the areas of environmental science, measurement science, soil science, bio-geochemistry, theoretical physics, alloy physics, magnetism, and planetary science.

My scientific h-index impact factor is 39 (84% of Nobel Prize winners in physics had h-indexes of at least 30), and my articles have been cited more than 5,000 times in peer-reviewed scientific journals. My publication record, citations statistics, and impact factors are publicly available at Google Scholar, at the URL https://scholar.google.ca/citations?user=1ChsRsQAAAAJ .

My recent non-committee-reviewed articles about the science of the COVID-19 epidemic and the science of masks for preventing viral respiratory diseases have been read more than 0.5 million times on *ResearchGate*, and more times on other venues. My recent video interviews and reporting videos about the science of COVID-19 and face masks have been viewed more than 1 million times.

My personal knowledge and ability to evaluate the facts in this article are grounded in my education, research, training and experience, as follows:

i. *Regarding environmental nanoparticles.* Viral respiratory diseases are transmitted by the smallest size-fraction of virion-laden aerosol particles, which are reactive environmental nanoparticles. Therefore, the chemical and physical stabilities and transport properties of these aerosol particles are the foundation of the dominant

contagion mechanism through air. My extensive work on reactive environmental nanoparticles is internationally recognized, and includes: precipitation and growth, surface reactivity, agglomeration, surface charging, phase transformation, settling and sedimentation, and reactive dissolution. In addition, I have taught the relevant fluid dynamics (air is a compressible fluid), and gravitational settling at the university level, and I have done industrial-application research on the technology of filtration (face masks are filters).

ii. *Regarding molecular science, molecular dynamics, and surface complexation.* I am an expert in molecular structures, reactions, and dynamics, including molecular complexation to biotic and abiotic surfaces. These processes are the basis of viral attachment, antigen attachment, molecular replication, attachment to mask fibers, particle charging, loss and growth in aerosol particles, and all such phenomena involved in viral transmission and infection, and in protection measures. I taught quantum mechanics at the advanced university level for many years, which is the fundamental theory of atoms, molecules and substances; and in my published research I developed X-ray diffraction theory and methodology for characterizing small material particles.

iii. *Regarding statistical analysis methods.* Statistical analysis of scientific studies, including robust error propagation analysis and robust estimates of bias, sets the limit of what reliably can be inferred from any observational study, including randomized controlled trials in medicine, and including field measurements during

epidemics. I am an expert in error analysis and statistical analysis of complex data, at the research level in many areas of science. Statistical analysis methods are the basis of medical research.

iv. *Regarding mathematical modelling.* Much of epidemiology is based on mathematical models of disease transmission and evolution in the population. I have research-level knowledge and experience with predictive and exploratory mathematical models and simulation methods. I have expert knowledge related to parameter uncertainties and parameter dependencies in such models. Recently, in collaboration, I have examined the instantaneous reproductive rate of COVID-19 infections in response to government masking impositions, in U.S. States.

v. *Regarding measurement methods.* In science there are five main categories of measurement methods: (1) spectroscopy (including nuclear, electronic and vibrational spectroscopies), (2) imaging (including optical and electron microscopies, and resonance imaging), (3) diffraction (including X-ray and neutron diffractions, used to elaborate molecular, defect and magnetic structures), (4) transport measurements (including reaction rates, energy transfers, and conductivities), and (5) physical property measurements (including specific density, thermal capacities, stress response, material fatigue…). I have taught these measurement methods in an interdisciplinary graduate course that I developed and gave to graduate (M.Sc. and Ph.D.) students of physics, biology, chemistry, geology, and engineering for many years. I have made fundamental discoveries and advances in areas of

spectroscopy, diffraction, magnetometry, and microscopy, which have been published in leading scientific journals and presented at international conferences. I know measurement science, the basis of all sciences, at the highest level.

It would be insufficient for me to be a simple medical doctor (MD) or public health officer. My relevant knowledge and ability stems from my broad multi-disciplinary knowledge, in light of the recognized difficulty of the question. For example, recently, 239 scientists put it this way:

> Understanding the transmission of respiratory infections indoors requires expertise in many distinctly different areas of science and engineering, including virology, aerosol physics, flow dynamics, exposure and epidemiology, medicine, and building engineering, to name the most significant. No one person has expertise in all these areas. However, collectively, the community of the signatories to the Comment understands the characteristics and mechanisms behind the generation of respiratory microdroplets, survival of viruses in the microdroplets, transport of the microdroplets and human exposure to them, and the airflow patterns that carry microdroplets in buildings. We have dedicated our careers working in this multidisciplinary field, and our statement stems from our collective expertise spanning the entire field.
>
> (First paragraph on page 1 of the Supplementary data, for: Morawska and Milton et al. (239 signatories) (6 July 2020) "**It is Time to Address Airborne Transmission of COVID-19**", in *Clinical Infectious Diseases*. [4])

Government responses have been a public-health and safety catastrophe

The forced masking laws are being recommended and enacted in a declared-pandemic context in which government responses to COVID have been disastrous, both in terms of response-induced deaths and permanent societal damage:

a. In my 2 June 2020 article "**All-cause mortality during COVID-19: No plague and a likely signature of mass homicide by government response**", I showed that an unnatural sharp "COVID-peak" in the all-cause mortality by week occurred across the world synchronously initiated by the 11 March 2020 WHO declaration of the pandemic and recommendation for States to empty their critical care units in preparation, which corresponded to a large acceleration of deaths of immunevulnerable elderly. [5]

b. Since my article, at least two published scientific papers have arrived at the same conclusion regarding accelerated or excess non-COVID-19 deaths occurring within the said "COVID-peak", as follows.

c. The 1 July 2020 article "**Excess Deaths From COVID-19 and Other Causes, March-April 2020**", by Woolf SH et al. in *JAMA* reports large numbers of said "COVID-peak" coincidence excess deaths actually caused by ●heart disease, ●diabetes, ●cerebrovascular disease, and ●Alzheimer disease, reported in their

Figure. [6] This means that the government responses caused these large numbers of non-COVID-19 excess deaths, unless one believes in supernatural coincidences.

d. The 2 July 2020 (date posted) article “**An Improved Measure of Deaths Due to COVID-19 in England and Wales**”, by Williams, S et al., available at *SSRN* reports that more than half of the deaths in the said “COVID-peak” are non-COVID-19 deaths, and concludes: [7]

> Three key findings from our empirical analysis are as follows. First, although it has been widely reported that COVID-19 has been highly concentrated in the elderly, we find that it has been particularly concentrated in the very elderly (75-84 and 85+ years), and less so in the 65-74 age category. Second, using two sets of COVID identifiers, we find from the beginning of the two periods when we assume the lockdown was having an impact, through to the end of our study period (week ending 17th or 24th April 2020 - week ending 8th May 2020), that our weekly estimates of COVID deaths for five cases (the total; the 75-84 and 85+ age categories; males; and females) diverge from the corresponding 5 year average excess deaths measure. Over these periods, we find that, on average per week, our estimates of COVID deaths for these five cases were (in absolute 6 terms) considerably below the corresponding 5 year average excess deaths measure. For example,on average per week, our estimate of total COVID deaths over these periods was lower than the corresponding 5 year average excess deaths measure by 4670-4727 deaths (54%-63%). For the above five cases, and in line with our hypothesis, we posit that the 5 year average excess deaths contains a large number of non-COVID deaths. Third, and relatedly, our analysis suggests that the UK’s lockdown has had a net positive impact on mortalities. That is to say, it resulted in more, not less, deaths.

e. This means that government responses in many jurisdictions caused more deaths than the virus itself.

f. The mechanism for the deaths caused by government response are manifold, and from my reading of the scientific and policy literature include:

- reduced access to care for chronic conditions,
- the direct impact of psychological stress,
- the practice of exporting ill patients from chronic care facilities to long-term care facilities, and
- the practice of locking in and isolating long-term care facility residents.

g. The direct impacts of fear and psychological stress on immunevulnerable elderly persons have most certainly been underestimated. Psychological stress is proven to be a factor that can measurably depress the immune system and induce diseases, including: immune response dysfunction, depression, cardiovascular disease and cancer: “**Psychological Stress and Disease**”, by Cohen, S et al., in *JAMA*. [8]

h. Furthermore, it is established since 1991 that psychological stress dramatically increases susceptibility to viral respiratory diseases, even in young healthy college-age subjects: “**Psychological Stress and Susceptibility to the Common Cold**”, by Cohen, S et al., in *The New England Journal of Medicine*. [9]

i. Additionally, it is known that social isolation increases susceptibility to viral respiratory diseases: “**Social ties and susceptibility to the common cold**”, by Cohen, S et al. in *JAMA*. [10]

j. Thus, government responses that induced fear, psychological stress, and isolation, including face masking impositions, were diametrically opposite to known science and had the predictable effect, given their scale, of directly in themselves causing large numbers of deaths.

k. This does not count the harm from restructuring the economy, corporate activity, and institutional networks. In a letter dated 19 May 2020, more than 500 USA physicians wrote to President Trump that “**In medical terms, the shutdown was a mass casualty incident.**” [11] In their letter, they concluded:

> The millions of casualties of a continued shutdown will be hiding in plain sight, but they will be called alcoholism, homelessness, suicide, heart attack, stroke, or kidney failure. In youths it will be called financial instability, unemployment, despair, drug addiction, unplanned pregnancies, poverty, and abuse.

l. There can be little doubt that governments have made fatal errors in responding to COVID-19, causing widespread harm and death.

m. Imposing face masks on the healthy general population is another such disastrous blunder:

- Repeated large randomized controlled trials (RCT) with verified outcome (lab-confirmed infection) and several systematic reviews of RCTs have proven that face masks have no detectable benefit for reducing the risk of person to person transmission of a viral respiratory disease.
- Recent laser visualization of simulated coughs has proven that cloth masks do not prevent exhalation of clouds of suspended aerosol particles, above, below and through the masks. [12]
- The known significant potential harms of face masks, and cloth face masks in particular, have neither been studied nor ruled out nor been the subject of harm mitigation trials.
- For example, home fabrics are hydrophilic, whereas medical masks are hydrophobic, the many harmful consequences of which have not been studied, and are virtually never mentioned.
- All-population face mask impositions increase fear and psychological stress.
- All-population face mask impositions cause:
    - widespread discomfort,
    - impaired breathing,
    - impaired vision (e.g., fogging of glasses),
    - impaired communication,
    - psychological social distancing,
    - skin irritation and infections,

- impaired self-expression,
- prolonged exposure to bacterial cultures near the eyes, nose and mouth,
- possible collection and delivery of viral pathogens that would otherwise not be inhaled, and
- possible amplification of the exhaled aerosol size-fraction of infectious particles.

The "growing body of evidence" mantra needs to stop

I gave my review of the scientific literature regarding the measured (in)efficacy of masks to reduce the risk of transmission of viral respiratory diseases in my article published on 11 April 2020 at *ResearchGate*, entitled "**Masks Don't Work: a Review of Science Relevant to Covid-19 Social Policy**". [13]

The said article [13] was read some 400 K times on *ResearchGate*, was published in several venues, and has been the subject of many commentary articles and interviews. It was critiqued by an incompetent academic and columnist at *Phycology Today,* who was spectacularly exposed in a live debate with me: "**Digi-Debates. The Face Mask Debate**", Digi Debates YouTube Channel, 25 July 2020, https://youtu.be/AQyLFdoeUNk , and see: https://www.digi-debates.com/ .

My conclusion in the said article [13] is that the policy-grade science of the recent decade conclusively shows that any benefit from masks is too small to be detected in trials designed to detect a benefit in this application.

My conclusions in the said article [13] regarding the RCT-with-verified-outcome studies are robust, and have again been corroborated by the very latest systematic reviews of RCTs, and by the most recently published expert assessments [14] [15] [16] [17] [18], as shown below.

In contrast, politicians of all jurisdictions, city mayors and local public health officers claim by mantra that this decade's worth of policy-grade research is being overturned by "emerging" evidence. Well, if it is "emerging", then it has not yet arrived.

Dr. Eileen de Villa, Medical Officer of Health, Toronto Public Health (TPH), announced her recommendation to the Toronto City Council on twitter as: Dr. Eileen de Villa @epdevilla "Since the beginning of this pandemic I've asked residents to take care of each other. Today I'm asking for this again & this is why I'm asking City Council to require masks or face covering in all public settings to help stop the spread of #COVID19: bit.ly/38cYlu8" 10:46 AM · June 30, 2020 · Twitter for iPhone.

The link provided in this tweet is to a TPH document (the "Recommendation") dated "June 30, 2020 at 9 a.m." entitled "**Update on COVID-19, Dr. Eileen de Villa, Medical Officer of Health**". [19]

The Recommendation contains ten (10) paragraphs as “bullets”. At the 2nd bullet, Dr. de Villa has “**there is a growing body of emerging evidence that shows that non-medical masks can help prevent the spread of COVID-19**”. This is squarely false. There is not a single published scientific study “that shows that non-medical masks can help prevent the spread of COVID-19”, let alone “a growing body”. In order to measure “the spread of COVID-19”, one has to actually measure “the spread of COVID-19”. In fact, there is a growing body solely of spin and of false statements about the scientific research literature. For comparison, see the sober recent Public Health Ontario (PHO) synopsis. [20]

As another of a multitude of such examples of the use of the said mantra, mayor Jim Watson of the City of Ottawa, Canada, in a well-crafted statement put it this way, in answering a recent demand by the Ontario Civil Liberties Association, while ignoring all the points raised by OCLA: [21] [22]

> “Increasing evidence supports wearing a mask when in enclosed public spaces as an important measure in reducingCOVID-19 transmission, while the risk of rising rates of infection continues. The scientific community and public health organizations around the world have concluded that the cumulative weight of evidence supports that face masks lessen the rates of transmission of COVID-19 from wearers. Most agree that face masks work best by reducing the amount of virus that is projected into the air in respiratory micro-droplets from someone who is infected with the virus. Additionally, other community level measures such as physical distancing and hand hygiene should continue to be employed to decrease transmission of COVID-19.
>
> While we respect that you may not necessarily agree with this public health initiative, we trust that you will understand the basis that prompted OPH to recommend that Council enact a by-law.”

Basically, the mayor is relying on “we are all saying it”.

Here is why “what they are all saying” is simply worthless. The new mantra is pure propaganda that is diametrically contrary to all the authoritative science reports, as follows:

a. In medical research, the only scientifically valid way to test a medical intervention, such as wearing a face mask or prescribing any preventative treatment, is to use the universally accepted comparative study (e.g., face mask versus no face mask) specifically designed to remove selection and observational bias from the study. This is called a “randomized controlled trial” (RCT).

b. Arguably the world’s leading medical standards and medical statistician expert, **Dr. Janus Christian Jakobsen**, author of the highly cited “Thresholds for statistical and clinical significance in systematic reviews with meta-analytic methods” (Jakobsen, JC et al., in *BMC Med Res Methodol* [23], has emphatically stated: [24]

> Clinical experience or observational studies should never be used as the sole basis for assessment of intervention effects — randomized clinical trials are always needed. Therefore, always randomize the first patient as Thomas C Chalmers suggested in 1977. Observational studies should primarily be used for quality control after treatments are included in clinical practice.

Abstracted Conclusion (p. 1) in: "**The Necessity of Randomized Clinical Trials**", by Jakobsen and Gluud, in the *British Journal of Medicine & Medical Research*. [24]

c. Meldrum in her "**A Brief History of the Randomized Controlled Trial: From Oranges and Lemons to the Gold Standard**" (Meldrum, Marcia L., in Hematology/Oncology Clinics of North America) [25], puts it this way (p. 746):

> Nevertheless, the RCT remains the "gold standard." Its power as a model for good practice rests on its imposition of experimental order on the clinical setting and its production of numerical results that may not be absolutely accurate but that are unquestionably precise. As Theodore Porter has argued, the value of the precise quantitative result is that it is readily translated outside its original experimental setting, for replication, comparison, and adaptation elsewhere.[ref]
>
> The inferential authority of the RCT has been such that it is accepted as a standard for "rational therapeutics" by physicians and regulatory authorities and also by patients and populations at risk.

d. It appears that "regulatory authorities" in Ontario, Canada, are not up to speed on modern medical-practice standards.

e. Recent medical history has shown that non-RCT comparative or observational studies can be egregiously wrong, with devastating negative public health consequences. Two examples are particularly well known, among many more:

(i) Non-RCT studies of the antiarrhythmic agents flecainide and encainide were glowing when the drugs were put onto the market in the late 1980s, then a RCT showed that these drugs increased mortality rather than had any benefit.

(ii) Decades of non-RCT "observational studies" were the basis for widespread hormone replacement therapy for post-menopausal women, until 2002 and later when published RCTs showed that these treatments actually increased myocardial infarctions (heart attacks) rather than decreased them as intended. The RCTs also found that the treatment increased the risk of incident breast cancer, which had not previously been detected in the decades of use. See: "**Risks and benefits of estrogen plus progestin in healthy postmenopausal women: principal results From the Women's Health Initiative randomized controlled trial**" (Writing Group for the Women's Health Initiative Investigators, in *JAMA*.) [26]

f. In my article "**Masks Don't Work: a Review of Science Relevant to Covid-19 Social Policy**" [13], I concluded (p. 4):

> No RCT study with verified outcome shows a benefit for HCW or community members in households to wearing a mask or respirator. There is no such study. There are no exceptions.
>
> Likewise, no study exists that shows a benefit from a broad policy to wear masks in public (more on this below).
>
> Furthermore, if there were any benefit to wearing a mask, because of the blocking power against droplets and aerosol particles, then there should be more benefit from wearing a respirator (N95) compared to a surgical mask, yet several large meta-analyses, and all the RCT, prove that there is no such relative benefit.

g. In my co-signed **21 June 2020 letter to the Executive Director of the WHO** [1], we (the Ontario Civil Liberties Association) put it this way:

> Second, more importantly, you fail to mention that several randomized controlled trials with verified outcomes (infections) were specifically designed to detect a benefit, and did not find any measurable benefit, for any viral respiratory disease. This includes the many randomized controlled trials that find no difference between open-sided surgical masks and respirators. [Footnote-2: citing and quoting from ten (10) scientific studies.]
>
> You failed to mention that such results set a probabilistic upper limit on mask effectiveness, and you failed to calculate this upper limit. Instead, you repeat the misleading notion that reliable evidence has “not yet” been found to confirm your adopted
> bias.
>
> In other words, if masks were even moderately effective at reducing the risk of infection, then a benefit would have been statistically detected in one or more of the many reliable trials that have already been made.
>
> More fundamentally, a major problem with your document is that you wrongly rely on substandard scientific reports as constituting usable “evidence”. With public policy, especially health policy having draconian consequences, there must be a standards threshold below which a given report cannot be used as an indicator of reality. The reason that science requires randomized controlled trials with verified outcomes is precisely because other study designs are susceptible to bias.
>
> The context of a new disease and of a publicized pandemic is one in which all reporting (media, political, and scientific) is susceptible to large bias. The mechanisms of the biases are well known and anticipated, such as: political posturing, partisan conflicts, career advancement, publication-record padding, “discovery” recognition, public-interest and public-support mining, institutional and personal reputational enhancement, funding opportunities, corporate interests, and so on.
>
> Group bias is not an uncommon phenomenon. Large numbers of bias-susceptible studies that agree are of little value. Any study that does not apply the established scientific tools for avoiding observational bias should be presumed to be biased, in any draconian policy context.

> That is why the WHO cannot collect and rely on potentially biased studies to make recommendations that can have devastating effects (see below) on the lives of literally billions. Rather, the WHO must apply a stringent standards threshold, and accept only randomized controlled trials with verified outcomes. In this application, the mere fact that several such quality studies have not ever confirmed the positive effects reported in bias-susceptible reports should be a red flag.
>
> For example, two amply promoted recent studies that do not satisfy the standards threshold, and that, in our opinion, have a palpable risk of large bias are the following. […]

h. My statements about the scientific evidence regarding masks are corroborated by all the concurrent and subsequent publications of leading experts on this question of reliable bias-free studies, as follows.

i. ***>>>*** "***Rapid Expert Consultation on the Effectiveness of Fabric Masks for the COVID-19 Pandemic***" (National Academies of Sciences, Engineering, and Medicine, 8 April 2020): [17]

> (p.2) In considering the evidence about the potential effectiveness of homemade fabric masks, it is important to bear in mind how a respiratory virus such as SARS-CoV-2 spreads from person to person. Current research supports the possibility that, in addition to being spread by respiratory droplets that one can see and feel, SARS-CoV-2 can also be spread by invisible droplets, as small as 5 microns (or micrometers), and by even smaller bioaerosol particles.[ref] Such tiny bioaerosol particles may be found in an infected person's normal exhalation.[ref] The relative contribution of each particle size in disease transmission is unknown.
>
> There is limited research on the efficacy of fabric masks for influenza and specifically for SARSCoV-2. As we describe below, the few available experimental studies have important limitations in their relevance and methods. Any type of mask will have its own capacity to arrest particles of different sizes. Even if the filtering capacity of a mask were well understood,

however, the degree to which it could in practice reduce disease spread depends on the unknown role of each particle size in transmission.

Asymptomatic but infected individuals are of special concern, and the particles they would emit from breathing are predominantly bioaerosols. […]

(p. 3) An additional consideration in the effectiveness of any mask is how well it fits the user.[ref] Even with the best material, if a mask does not fit, virus-containing particles can escape through creases and gaps between the mask and face. Leakage can also occur if the holding mechanism (e.g., straps, Velcro®) is weak. We found no studies of non-expert individuals' ability to produce properly fitting masks. Nor did we find any studies of the effectiveness of masks produced by professionals, when following instructions available to the general public (e.g., online). […]

(p. 6) **CONCLUSIONS** […] The current level of benefit, if any, is not possible to assess.

j. **>>>** "**Nonpharmaceutical Measures for Pandemic Influenza in Nonhealthcare Settings—Personal Protective and Environmental Measures**" (Xiao, J et al., in *Emerging Infectious Diseases*, 5 May 2020): [14]

(p. 967: Abstract) Although mechanistic studies support the potential effect of hand hygiene or face masks, evidence from 14 randomized controlled trials of these measures did not support a substantial effect on transmission of laboratory-confirmed influenza. We similarly found limited evidence on the effectiveness of improved hygiene and environmental cleaning. We identified several major knowledge gaps requiring further research, most fundamentally an improved characterization of the modes of person-to-person transmission.

k. **>>>** "**Masks for prevention of viral respiratory infections among health care workers and the public: PEER umbrella systematic review**" (Dugré et al., in *Canadian Family Physician*, July 2020): [15]

(p. 509, Abstract) **Synthesis** In total, 11 systematic reviews were included and 18 RCTs of 26 444 participants were found, 12 in the community and 6 in health care workers. Included studies had limitations and were deemed at high risk of bias. Overall, the use of masks in the community did not reduce the risk of influenza, confirmed viral respiratory infection, influenzalike illness, or any clinical respiratory infection. […]

Conclusion This systematic review found limited evidence that the use of masks might reduce the risk of viral respiratory infections. […]

l. **>>>** Moe et al. summarized the detailed study of Dugré et al. [15] in their praxis article for medical practitioners: "**PEER simplified tool: mask use by the general public and by health care workers**" (Moe et al., in *Canadian Family Physician*, July 2020) [16]. Their Figure 1 (p. 506) has:

MASKS FOR THE GENERAL PUBLIC

Based on evidence from randomized controlled trials

If I wear a surgical mask while out in public, will it protect me from flu-like illness?

- 2 trials 1683 people
- The reduction in flu-like illness may be 4% (range: 0-8%) over 6 weeks.
- But no difference in lab-confirmed influenza

What about wearing a surgical mask at home after a household member becomes sick?

- Sick person wears mask: 2 trials, 903 people
- Healthy household members wear masks: 1 trial, 290 people
- Healthy and sick people wear masks: 4 trials, 2750 people
- In all three scenarios, wearing a mask did NOT reduce the risk of getting flu-like illness or confirmed influenza.

m. Here, note that, as always, "flu-like illness" or "influenza-like illness" (ILI) means non-laboratory-confirmed infection, based on reported symptoms or clinical observation. Such determinations are not "verified outcomes" and are thus more susceptible to bias.

n. **>>>** "**Masking lack of evidence with politics**" (Jefferson and Heneghan, in *Centre for Evidence Based Medicine (CEBM)*, Oxford University, 23 July 2020): [18]

> (p. 1) The increasing polarised and politicised views [ref] on whether to wear masks in public during the current COVID-19 crisis hides a bitter truth on the state of contemporary research and the value we pose on clinical evidence to guide our decisions.
>
> In 2010, at the end of the last influenza pandemic, there were six published randomised controlled trials with 4,147 participants focusing on the benefits of different types of masks.[ref] Two were done in healthcare workers and four in family or student clusters. The face mask trials for influenza-like illness (ILI) reported poor compliance, rarely reported harms and revealed the pressing need for future trials.
>
> Despite the clear requirement to carry out further large, pragmatic trials a decade later, only six had been published: five in healthcare workers and one in pilgrims.[ref] This recent crop of trials added 9,112 participants to the total randomised denominator of 13,259 and showed that masks alone have no significant effect in interrupting the spread of ILI or influenza in the general population, nor in healthcare workers.
>
> (p. 2) What do scientists do in the face of uncertainty on the value of global interventions? Usually, they seek an answer with adequately designed and swiftly implemented clinical studies as has been partly achieved with pharmaceuticals. We consider it is unwise to infer causation based on regional geographical observations as several proponents of masks have done. Spikes in cases can easily refute correlations, compliance with masks and other measures is often variable, and confounders cannot be accounted for in such observational research. […]
>
> The small number of trials and lateness in the pandemic cycle is unlikely to give us reasonably clear answers and guide decision-makers. This abandonment of the scientific modus operandi and lack of foresight has left the field wide open for the play of opinions, radical views and political influence.

So, what actually is the “growing body of evidence”?

Given the above-documented contradiction between the claimed “growing body of evidence” and the actual “all RCTs say the opposite of what is claimed”, one can reasonably ask: What are Ontario public health officers thinking of when they assert “there is a growing body of emerging evidence that shows that non-medical masks can help prevent the spread of COVID-19”?

One answer comes from the Simcoe-Muskoka District Health Unit (Ontario, Canada) webpage entitled “FAQ’s- Wearing a Face Covering in Indoor Public Spaces”, updated 24 July 2020. The latter webpage has the section: [27]

> **What is the evidence that supports the use of masks?**
>
> There is a growing body of scientific evidence that indicates the widespread use of face coverings by all persons decreases the spread of respiratory droplets. Public health experts also support the widespread use of face coverings to decrease transmission of COVID-19.
>
> At this link you will find a collection of expert opinions and studies on face coverings. This list is for informational purposes only and is not representative of all articles and studies available on the subject, nor does this list cover all articles and studies that are reviewed by our staff and our Medical Officer of Health.

The said “link” is to a webpage of the Wellington-Dufferin-Guelph public health unit, entitled “Your Health / COVID-19 Information for the Public / Reliable Information Sources”, accessed on 28 July 2020. [28] The latter webpage has a section entitled

“EXPERT OPINIONS”, having eight (8) entries, and a section entitled “EVIDENCE AND STUDIES ON FACE COVERINGS (UPDATED ON JULY 23)”, having thirty (30) entries.

The eight (8) so-called “expert opinions” are merely “op-ed” type commentaries not providing any new data, evidence, or perspectives. These do not constitute “a growing body of emerging evidence”, nor do they add any evidence whatsoever.

The thirty (30) so-called “evidence and studies” (ES) can be described as follows, numbering them ES-1 through ES-30 in the order given (alphabetical order of first-author):

ES-1 through ES-30: None of these studies are RCTs, irrespective of whether any outcomes (infections) are “verified” (lab-confirmed) or not. Some are actually “op-ed” style opinions. Some are tentative modelling studies. Some are population studies. Some are physical mask-filtering studies. A few are overview reports. A few purport to be “meta-analyses” or “systematic reviews” of old RCT and non-RCT studies (see below). None can be considered additions to “a growing body of emerging evidence”, at least not usable policy-grade evidence. All are susceptible to large bias.

ES-1: “Alberta Health Services COVID-19 Scientific Advisory Group. **Rapid Response Report: What is the effectiveness of wearing medical masks, including home-made masks, to reduce the spread of COVID-19 in the community?** Updated 2020 June.” [29] ⟶

The first two bullets in the section entitled "Key Messages from the Evidence Summary" are (page 1):

> • As medical masks are often bundled with other IPC interventions and have variable compliance, clinical trials on the effectiveness of medical masks have been challenging. Systematic reviews of randomized controlled trials in health care settings have not demonstrated a significant reduction in acute respiratory infections, (ARIs), ILIs or laboratory confirmed viral infections with medical mask use although it is acknowledged there were methodological flaws and smaller underpowered studies in the data analyzed.
> • There is a paucity of clinical evidence in favor of using medical masks in the community, with multiple randomized trials demonstrating mixed results which when pooled demonstrate no significant reduction in acute respiratory infections (ARIs), ILIs or laboratory confirmed viral infections. There are some lower quality studies showing a reduction in viral infection rates in households, in transmission of viral respiratory infections in the context of mass gatherings, and in university residences when combined with hand hygiene interventions.

The third-last bullet is:

> • There is limited evidence of harms related to community mask wearing with no studies identified that have systematically looked at potential harms. Such harms could include behavioral modifications such as risk compensation/non-adherence to social distancing or optimal hand hygiene practices, self-contamination, induction of facial rashes, and increasing real or perceived breathing difficulties. There are also concerns about poor compliance or tolerance of masks in children or those with cognitive challenges and communication difficulties.

The last bullet is:

> • Pre-symptomatic transmission and asymptomatic transmission of SARS-CoV-2 have been described but the degree to which they contribute to community spread is unclear, At this point, there is no direct evidence that the use of a medical or homemade cloth mask or the wider use of masks in the community significantly reduces this risk. For more information, refer to the Asymptomatic Transmission of SARS-CoV-2 rapid review.

ES-7: “Chu DK, Akl EA, Duda S et al. **Physical distancing, face masks, and eye protection to prevent person-to-person transmission of SARS-CoV-2 and COVID-19: a systematic review and meta-analysis**. *Lancet.* 2020 [30] ⟶

The DK Chu article has many problems. It was described in our letter to the WHO [1] as (pp. 5-6):

> The Chu study was funded by the WHO. It contains no randomized controlled trials, but rather uses a hodgepodge of data about associations of ill-defined factors. DK Chu et al.’s own appraisal of “certainty” regarding their conclusion about masks is “LOW” meaning “our confidence in the effect estimate is limited; the true effect could be substantially different from the estimate of the effect” (their Table 2), yet such a result is a basis for your recommendation to governments.

ES-18: “Liang M, Gao L, Cheng C, et al. **Efficacy of face mask in preventing respiratory virus transmission: a systematic review and meta-analysis**. *Travel Med Infect Dis.* 2020 May 28.” [31] ⟶

The Liang study purports to be a systematic review and meta-analysis yet it does not apply **PRISMA-P [Preferred reporting items for systematic review and meta-analysis protocols]** [32], nor does it perform **GRADE [Grading of Recommendations, Assessment, Development and Evaluations] reliability analysis**] [33], which are the established standard in such medical research intended to be used for policy guidance. If Liang did apply GRADE, it would fail, because its included studies are mostly non-RCT “case-control studies”, and because its confidence intervals encompass outcomes leading to the oppose recommendation of masks:

“**GRADE guidelines 6. Rating the quality of evidence—imprecision**, by Guyatta et al., in *Journal of Clinical Epidemiology*. [34]

ES-21: “MacIntyre CR, Chughtai AA. **A rapid systematic review of the efficacy of face masks and respirators against coronaviruses and other respiratory transmissible viruses for the community, healthcare workers and sick patients**. *Int J Nurs Stud.* 2020. [35] ⟶

The co-authors, MacIntyre and Chughtai, have both worked for or with 3M (a major proprietary mask and respirator manufacturer) and now work together; as they admit in the required “Conflict of Interest” statement. MacIntyre has made an industry or writing spin-laden articles about masks in scientific journals, which repeatedly have recast old RCT studies. This is one more in that pattern.

The authors MacIntyre and Chughtai claim “Results were reported according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) criteria (Moher et al., 2015).” (their “2. Methods” section, last sentence). In fact, this is false. The following numbered directives of PRISMA were not followed by MacIntyre and Chughtai (Table 3, [32]):

> #13 List and define all outcomes for which data will be sought, including prioritization of main and additional outcomes, with rationale
> #14 Describe anticipated methods for assessing risk of bias of individual studies, including whether this will be done at the outcome or study level, or both; state how this information will be used in data synthesis
> #15a Describe criteria under which study data will be quantitatively synthesized
> #15b If data are appropriate for quantitative synthesis, describe planned summary measures, methods of handling data, and methods of combining data from studies, including any planned exploration of consistency (e.g., I2, Kendall’s tau)

#15c Describe any proposed additional analyses (e.g., sensitivity or subgroup analyses, meta-regression)
#15d If quantitative synthesis is not appropriate, describe the type of summary planned
#16 Specify any planned assessment of meta-bias(es) (e.g., publication bias across studies, selective reporting within studies)
#17 Describe how the strength of the body of evidence will be assessed (e.g., GRADE)

Not having introduced one iota of new evidence, MacIntyre and Chughtai conclude (p. 5):

> In summary, there is a growing body of evidence supporting all three indications for respiratory protection – community, healthcare workers and sick patients (source control).

The work of MacIntyre and Chughtai is not science that can be used to guide public policy. It is substandard and misleading.

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All-cause mortality during COVID-19: No plague and a likely signature of mass homicide by government response

Denis G. Rancourt, PhD
Researcher, Ontario Civil Liberties Association (ocla.ca)

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Summary / Abstract

The latest data of all-cause mortality by week does not show a winter-burden mortality that is statistically larger than for past winters. There was no plague. However, a sharp "COVID peak" is present in the data, for several jurisdictions in Europe and the USA.

This all-cause-mortality "COVID peak" has unique characteristics:

- Its sharpness, with a full-width at half-maximum of only approximately 4 weeks;
- Its lateness in the infectious-season cycle, surging after week-11 of 2020, which is unprecedented for any large sharp-peak feature;
- The synchronicity of the onset of its surge, across continents, and immediately following the WHO declaration of the pandemic; and
- Its USA state-to-state absence or presence for the same viral ecology on the same territory, being correlated with nursing home events and government actions rather than any known viral strain discernment.

These "COVID peak" characteristics, and a review of the epidemiological history, and of relevant knowledge about viral respiratory diseases, lead me to postulate that the "COVID peak" results from an accelerated mass homicide of immune-vulnerable individuals, and individuals made more immune-vulnerable, by government and institutional actions, rather than being an epidemiological signature of a novel virus, irrespective of the degree to which the virus is novel from the perspective of viral speciation.

The paper is organized into the following sections:

- Cause-of-death-attribution data is intrinsically unreliable
- Year-to-year winter-burden mortality in mid-latitude nations is robustly regular
- Why is the winter-burden pattern of mortality so regular and persistent?
- A simple model of viral respiratory disease *de facto* virulence
- All-cause mortality analysis of COVID-19
- Interpreting the all-cause mortality "COVID peak"

Cause-of-death-attribution data is intrinsically unreliable

Assignment of cause of death, with infectious diseases and comorbidity, is not only technically difficult (e.g., Simonsen et al., 1997; Marti-Soler et al., 2014) but also contaminated by physician-bias, politics and news media.

This has been known since modern epidemiology was first practiced. Here is Langmuir (1976) quoting the renowned pioneer William Farr, regarding the influenza epidemic of 1847:

> Farr uses this epidemic to chide physicians mildly on their narrow views pointing out that sharp increases were observed not only in influenza itself but in bronchitis, pneumonia and asthma and many other non-respiratory causes, he states:
>
> > '… there is a strong disposition among some English practitioners not only to localize disease but to see nothing but the local disease. Hence, although it is certain that the high mortality on record was the immediate result of the epidemic of influenza, the deaths referred to that cause are only 1,157.'

And, such bias is generally recognized by leading epidemiologists (Lui and Kendal, 1987):

> … the decision to classify deaths into "pneumonia and influenza" is subjective and potentially inconsistent. On one hand, the effect of influenza or influenza-related pneumonia may be underestimated because underlying chronic diseases, particularly in the elderly, are usually noted as the cause of death on the death certificate. On the other hand, after influenza activity has been publicly reported there may be an increased tendency to classify deaths as due to "pneumonia and influenza," thereby amplifying the rate of increase in P&I deaths or, when a decline in influenza activity is reported, a bias toward decreasing the classification of deaths related to "pneumonia and influenza" may result. Surveys to evaluate these possibilities have not been done.

One can reasonably expect that in the current world of social media, with a World-Health-Organization-declared (WHO-declared) “pandemic”, such bias will only be greater compared to its presence in past viral respiratory disease epidemics.

For example, it is difficult to interpret the synchronicity of the WHO declaration of COVID-19 as a pandemic and the onset of the observed surge in reported COVID-19 cases and deaths as being the product of either coincidence or extraordinary forecasting ability of the global health-monitoring system:

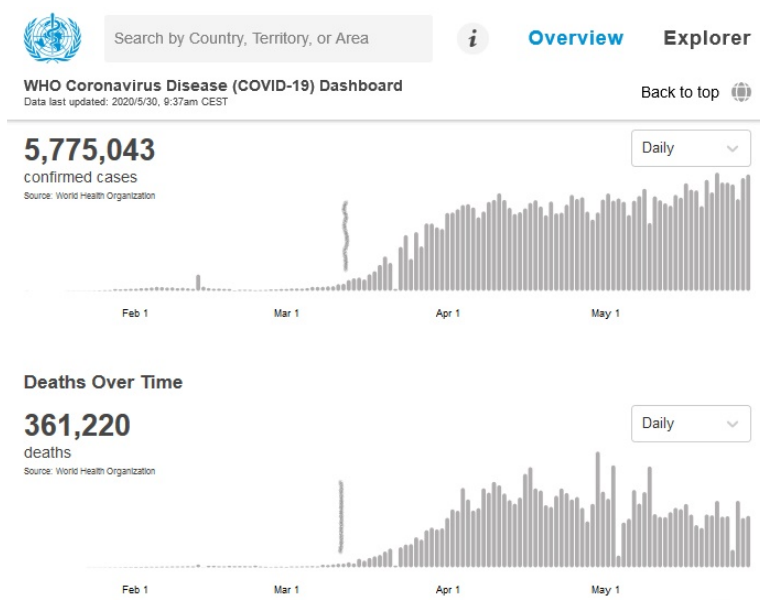


Figure 1: Globally reported COVID-19 cases, and reported COVID-19-assigned deaths, by day. WHO data was accessed on 30 May 2020. The vertical lines in pencil indicate the date at which the WHO declared the pandemic.

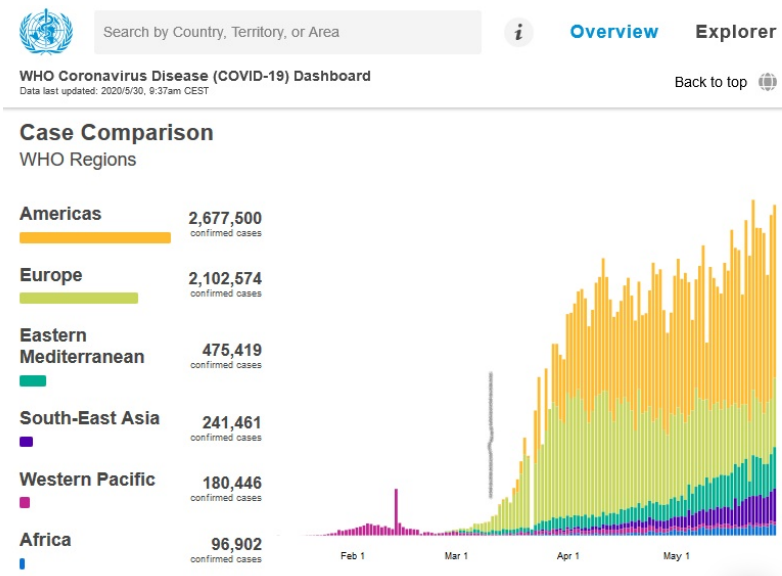


Figure 2: Globally reported new COVID-19 cases per day, discerning the continents. WHO data was accessed on 30 May 2020. The vertical line in pencil indicates the date at which the WHO declared the pandemic.

Instead, in light of past epidemics, it is more likely that this remarkable synchronicity phenomenon arises from biased reporting, in the flexible context of using urgently manufactured laboratory tests that are not validated, clinical assessments of a generic array of symptoms, and tentative cause-of-death assignations of complex comorbidity circumstances.

That is why rigorous epidemiological studies rely instead on all-cause mortality data, which cannot be altered by observational or reporting bias (as discussed in Simonsen et al., 1997; and see Marti-Soler et al., 2014). A death is a death is a death.

Year-to-year winter-burden mortality in mid-latitude nations is robustly regular

Modern human mortality in mid-latitude temperate-climate regions is robustly seasonal. Graphs of number of all-cause deaths per unit of time (month, week, day), in given regions, have a yearly pattern, with a peak-to-trough amplitude of typically 10% to 30% of the trough-baseline value, largely irrespective of the specific pathogens that populate the specific seasons. High mortality occurs in winter, and is thus inverted in the Northern and Southern hemispheres (e.g., Marti-Soler et al., 2014).

For the USA, the phenomenon is well illustrated in this figure from Simonsen et al. (1997):

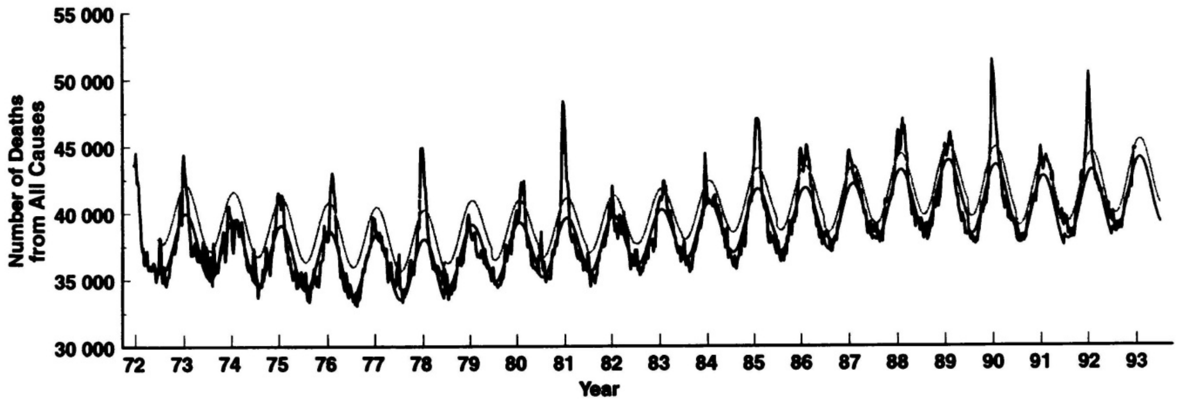


Figure 3: All-cause mortality, by week, for the USA, 1972 to 1993 (Simonsen et al., 1997; from their Fig. 1).

In such a graph, the area under a peak, to its trough-level baseline, is the total number of yearly winter-burden deaths above the trough baseline. The thus calculated yearly “excess” number of deaths, here (in the era 1972-1993), is always approximately 8% to 11% of the total yearly trough-baseline-level deaths, also approximately 8% to 11% of the yearly all-cause mortality.

This regular and seasonal “excess” mortality, or winter burden, has been an epidemiological challenge to understand, although, starting with Farr, many epidemiologists originally attributed it almost entirely to the seasonal influenza-like viral respiratory diseases.

Nonetheless, the agonizing difficulty to understand the cause(s) of this remarkably regular and global (both hemispheres, but inverted) pattern persists, as illustrated in the words of Marti-Soler et al. (2014) (references omitted):

> Given that mortality from cancer showed virtually no seasonality pattern, the seasonality of overall mortality is driven mostly by seasonality of both CVD [cardiovascular diseases] and non-CVD/non-cancer mortality. For these conditions, and particularly for CVD, exposure to cold is a plausible explanation for the observed seasonality, given relationship of cold climate with latitude. Several longitudinal studies have demonstrated that a decrease in outdoor temperature was associated with a rise in all cause mortality. However, other latitude-dependent factors, such as dietary habits, sun exposure (vitamin D levels) and human parasitic and infectious agents might also play a role. The magnitude of the seasonal pattern for CVD mortality was highest than that for all cause mortality. The seasonality of CVD mortality might be partly due to the joint seasonality of several known CVD risk factors, as described previously. Similarly, lifestyle factors such as diet and physical activity also tend to differ during summer and winter months. Moreover, exposure to cold increases energy expenditure, peripheral vasoconstriction and cardiac afterload, thus potentially triggering myocardial ischemia

and stroke. Finally, winter prone influenza infection might also be a trigger for CVD deaths by exacerbating CVD conditions or due to secondary complications. This is likely to be the case of concentration of air pollutants.

The seasonality of non-CVD/non-cancer mortality can relate to the facts that chronic obstructive pulmonary disease and pneumonia are frequent diseases in this category and that these disease are exacerbated by influenza, other influenza-like infections and concentrations of air pollutants, which are all more frequent in winter. A few other diseases in the non-CVD/non-cancer category also present a seasonal pattern, e.g. depression, suicide, and oesophageal variceal bleeding.

Why is the winter-burden pattern of mortality so regular and persistent?

Even the seasonality of the pneumonia and influenza ("P&I") part *alone* (which is a large part of what Marti-Soler et al. quantify as "non-CVD/non-cancer mortality") was not understood until a decade ago. Until recently, it was debated whether the P&I yearly pattern arose primarily because of seasonal change in virulence of the pathogens, or because of seasonal change in susceptibility of the host (such as from dry air causing tissue irritation, or diminished daylight causing vitamin deficiency or hormonal stress). For example, see Dowell (2001). In a sense, the answer is "neither".

In a landmark study, Shaman et al. (2010) showed that the seasonal pattern of respiratory-disease (P&I) excess mortality can be explained quantitatively on the sole basis of absolute humidity, and its direct controlling impact on transmission of airborne pathogens.

Lowen et al. (2007) demonstrated the phenomenon of humidity-dependent airborne-virus contagiousness in actual disease transmission between guinea pigs, and discussed potential underlying mechanisms for the measured controlling effect of humidity.

The underlying mechanism is that the pathogen-laden aerosol particles or aerosol-size droplets are neutralized within a half-life that monotonically and significantly decreases with increasing ambient absolute humidity. This is based on the seminal work of Harper (1961). Harper experimentally showed that viral-pathogen-carrying droplets were inactivated within shorter and shorter times, as ambient absolute humidity was increased.

Harper argued that the viruses themselves were made inoperative by the humidity ("viable decay"), however, he admitted that the effect could be from humidity-enhanced physical removal or gravitational sedimentation of the droplets ("physical loss"): "Aerosol viabilities reported in this paper are based on the ratio of virus titre to radioactive count in suspension and cloud samples, and can be criticized on the ground that test and tracer materials were not physically identical."

The latter ("physical loss") seems more plausible to me, since absolute humidity would have a universal physical effect of causing particle/droplet growth-by-condensation and gravitational sedimentation (and, conversely, loss-by-evaporation and aerosolization), and all tested viral pathogens have essentially the same humidity-driven "decay". Furthermore, it is difficult to understand how a virion (of any virus type) in a droplet would be molecularly or structurally attacked or damaged by an increase in ambient humidity. A "virion" is the complete, infective form of a virus outside a host cell, with a core of RNA or DNA and a capsid. No actual molecular or other mechanism of the humidity-driven intra-droplet "viable decay" of a virion postulated by Harper (1961) has, to date, been explained or studied, whereas gravitational sedimentation ("physical loss") is well understood.

In any case, the explanation and model of Shaman et al. (2010) is not dependant on the particular mechanism of the absolute-humidity-driven decay of virions in aerosol/droplets. Shaman's quantitatively demonstrated model of seasonal regional viral epidemiology is valid for either mechanism (or combination of mechanisms), whether "viable decay" or "physical loss".

The breakthrough achieved by Shaman et al. is not merely some academic point. Rather, it has profound health-policy implications, which have been entirely ignored or overlooked in the current coronavirus pandemic:

- It means that the seasonality of P&I mortality is directly driven by absolute-humidity-controlled contagiousness of the viral respiratory diseases.

If my view of the mechanism is correct (i.e., "physical loss" rather than "viable decay"), then:

- It additionally implies that the transmission vector must be small aerosol particles in fluid suspension in air, breathed deeply into the lungs, indoors; not hypothesized routs such as actual fluid or fomite contact, and not large droplets and spit (that are quickly gravitationally removed from the air, or captured in the mouth and digestive system).
- And it means that social distancing, masks, and hand washing can have little effect in the actual epidemic spread during the winter season (see: Rancourt, 2020).

On the epidemiology modelling side, Shaman's work implies that, rather than being a fixed number (dependent solely on the spatial-temporal structure of social interactions in a completely and variably susceptible population, and on the viral strain), the epidemic's basic reproduction number (R0) is predominantly dependent on ambient absolute humidity. For a definition of R0, see HealthKnowlege-UK (2020): R0 is "the average number of secondary infections produced by a typical case of an infection in a population where everyone is susceptible."

Shaman et al. showed that R0 must be understood to vary seasonally between humid-summer values of just larger than "1" and dry-winter values typically as large as "4" (for example, see their Table 2). In other words, the seasonal infectious viral respiratory diseases that plague temperate-climate regions every year go from being intrinsically mildly contagious to virulently

contagious, due simply to the bio-physical mode of transmission controlled by atmospheric absolute humidity, largely irrespective of any other consideration.

Furthermore, indoor airborne virus concentrations have been shown to exist (in day-care facilities, health centres, and onboard airplanes) primarily as aerosol particles of diameters *smaller than* 2.5 μm, such as in the work of Yang et al. (2011):

> "Half of the 16 samples were positive, and their total virus concentrations ranged from 5800 to 37 000 genome copies m^{-3}. On average, 64 per cent of the viral genome copies were associated with fine particles smaller than 2.5 μm, which can remain suspended for hours. Modelling of virus concentrations indoors suggested a source strength of $1.6 \pm 1.2 \times 10^5$ genome copies m^{-3} air h^{-1} and a deposition flux onto surfaces of 13 ± 7 genome copies m^{-2} h^{-1} by Brownian motion. Over 1 hour, the inhalation dose was estimated to be 30 ± 18 median tissue culture infectious dose ($TCID_{50}$), adequate to induce infection. These results provide quantitative support for the idea that the aerosol route could be an important mode of influenza transmission."

Such small particles (*smaller than* 2.5 μm) are part of air fluidity, are not subject to gravitational sedimentation, and can therefore be breathed deeply into the lungs.

The next question is: How many such pathogen-laden particles are needed to cause infection in a person of average immune-response capacity?

Yezli and Otter (2011), in their review of the minimal infective dose (MID), point out relevant features:

- most respiratory viruses are as infective in humans as in tissue culture having optimal laboratory susceptibility
- the 50%-probability MID ("$TCID_{50}$") has variably been found to be in the range 100−1000 virions
- there are typically 10^3–10^7 virions per aerolized influenza droplet with diameter 1 μm − 10 μm
- the 50%-probability MID easily fits into a single (one) aerolized droplet

For further background:

- A classic description of dose-response assessment is provided by Haas (1993).
- Zwart et al. (2009) provided the first laboratory proof, in a virus-insect system, that the action of a single virion can be sufficient to cause disease.
- Baccam et al. (2006) calculated from empirical data that, with influenza A in humans, "we estimate that after a delay of ~6 h, infected cells begin producing influenza virus

and continue to do so for ~5 h. The average lifetime of infected cells is ~11 h, and the half-life of free infectious virus is ~3 h. We calculated the [in-body] basic reproductive number, R_0, which indicated that a single infected cell could produce ~22 new productive infections."

- Brooke et al. (2013) showed that, contrary to prior modeling assumptions, although not all influenza-A-infected cells in the human body produce infectious progeny (virions), nonetheless, 90% of infected cell are significantly impacted, rather than simply surviving unharmed.

The above review means that all the viral respiratory diseases that seasonally plague temporal-climate populations every year are extremely contagious for two reasons: (1) they are transmitted by small aerosol particles that are part of the fluid air and fill virtually all enclosed air spaces occupied by humans, and (2) a single such aerosol particle carries the minimal infective dose (MID) sufficient to cause infection in a person, if breathed into the lungs, where the infection is initiated.

This is why the pattern of all-cause mortality is so robustly stable and distributed globally, if we admit that the majority of the burden is induced by viral respiratory diseases, while being relatively insensitive to the particular seasonal viral ecology for this operational class of viruses. This also explains why the pattern is inverted between the Northern and Southern hemispheres, irrespective of tourist and business air travel and so one.

Virologists and geneticists see viral strains, mutations, and species (Alimpiev, 2019), like a man with a hammer sees nails. Likewise, there are professional rewards for identifying new viral pathogens and describing new diseases. For these reasons, scientists have not seen the forest for the trees.

But the data shows that there is a persistent and regular pattern of winter-burden mortality that is independent of the details, and that has a well constrained distribution of year to year number of excess deaths (approximately 8% to 11% of the total yearly mortality, in the USA, 1972 through 1993). Despite all the talk of epidemics and pandemics and novel viruses, the pattern is robustly constant.

An anomaly worthy of panic, and of harmful global socio-economic engineering, would need to consist of a naturally caused yearly winter-burden mortality that is statistically greater than the norm. That has not occurred since the unique flu pandemic of 1918 (Hsieh et al., 2006).

The three recent epidemics assigned as pandemics, the H2N2 pandemic of 1957, the H3N2 pandemic of 1968, and the H1N1 pandemic of 2009, were not more virulent (in terms of yearly winter-burden mortality) than the regular seasonal epidemics (Viboud et al., 2010; Viboud et al., 2006; Viboud et al., 2005). In fact, the epidemic of 1951 was concluded to be more deadly, on the basis of P&I data, in England, Wales and Canada, than the pandemics of 1957 and 1968 (Viboud et al., 2006).

A simple model of viral respiratory disease *de facto* virulence

In the face of the persistent and regular pattern of winter-burden mortality, one is tempted to propose that the specific (structural, molecular, and binding) properties of the particular respiratory disease viral pathogen are not as determinative of mortality as virologists suggest. Instead, it is possible that mortality, in a given population exposed to these highly contagious viral pathogens that invade the lungs, is predominantly controlled by the population's distribution of immune-system capacity and preparedness.

A viral load enters the lungs. Once the viral antigen is recognized, an immune response is mounted.[1] A dynamic "war" ensues between the virus reproducing and spreading by infecting cells on the lining of the lungs, and the immune system doing everything it can to identify, locate and destroy infected cells before the said infected cells successfully can be productive of the virus.

The immune response is extraordinarily demanding of the body's metabolic energy resources (which is why you "feed a cold", "rest", and "stay warm"). The demand in metabolic energy is prioritized, and can compete with the demands of essential bodily functions and immune responses to other pathogens. This is why individuals with "aging" diseases and comorbidity conditions are particularly at risk: their rate of metabolic energy supply to the immune-system is limited by their co-conditions, and the demand is not met at a sufficiently high rate to win the "war". See: Straub (2017); Bajgar et al. (2015).

In a simple view of the infection (which I propose for illustration), a given individual, having a given state of health, can only provide metabolic energy to the immune system up to some maximum rate of supply, during the crucial stage of the "war". Call this "rate of energy supply for the immune response": RS. RS is in units of energy per unit time, J/s, or calories per second. If RS is sufficient to "win the war", and is sustained long enough, then the individual recovers from the infection, and the immune system stores a molecular memory of the viral antigen, which greatly reduces energy demand for future immune responses to attacks from the same or sufficiently similar virus. If RS is insufficient then the individual succumbs to the virus and dies.

Therefore, the seasonal virus can be characterized as having a virus-specific value of RS, RSv, which is the RS threshold for survival of the infected person. If RS > RSv, then the person recovers. If RS < RSv, then the person dies. The larger the RSv, the more virulent is the virus, and vice versa.

[1] See: "The immune system: Cells, tissues, function, and disease", medically reviewed by Daniel Murrell, MD on January 11, 2018 — Written by Tim Newman, at *medicalnewstoday.com*, accessed on 1 June, 2020. https://www.medicalnewstoday.com/articles/320101

A given human population (national or regional) will have a given distribution of RS values associated with the individual members of the population.

Mathematically, this distribution can be represented as a probability density of RS values. A probability-density value has units of number of persons per unit interval of RS. The total area under the probability density curve is the population, of the nation or region.

Figure 4 illustrates three hypothetical distributions of RS values, in three different populations of equal size. Here: “Germany” (solid-blue line) is for a current Western population, not having a particularly large elderly population; “Italy” (dashed-blue line) is for a current Western population having a large elderly population; and “Stressed” (solid-red line) is for a population of individuals subjected to high metabolic (or health) stress, such as might have been the case in 1918 England.

Such health stress can arise from nutritional deficiency, essential nutrient or vitamin efficiency, high levels of environmental stressor-agents, toxins, or pathogens, shelter deficiency (“fuel poverty”), oppressive working conditions, social-dominance oppression, substance abuse causing organ damage, and so on. There is a vast literature on these factors. As one anchor point, see: Sapolsky (2015); Sapolsky (2005).

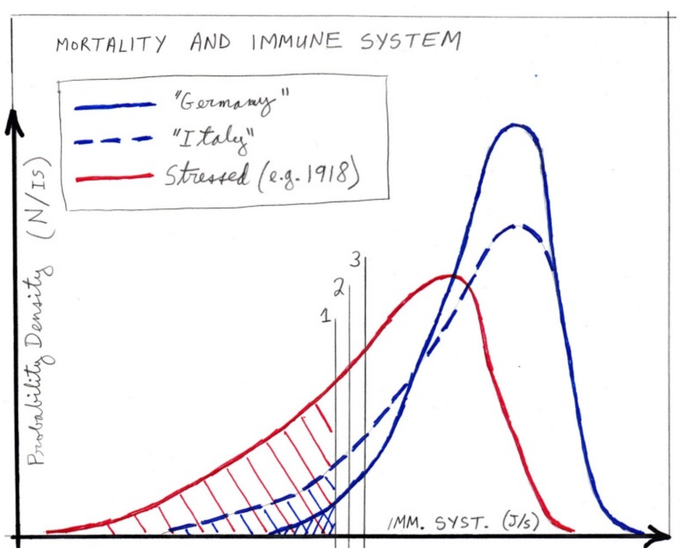


Figure 4: Probability densities of RS values, for three populations of equal size but differing in health-stress levels and health vulnerabilities, as explained in the text. The three vertical lines, drawn in pencil and labelled “1”, “2” and “3”, show three different virus-specific values of RSv, as explained in the text. The hatched areas are the fractions (of total area) representing the mortality fractions for the less virulent virus having RSv value labelled “1”.

In this model, therefore, comparative mortality between populations, for a given viral pathogen, is determined by the different health states (distributions of RS values of the individuals) of the compared infected populations.

This is for the full cycle of infection and recovery. It says little about both the death rates on a daily basis and age distributions, which depend on the natural or forced spread of the infection, which in turn is not necessarily uniform in time and space but rather can target particular segments of the population, such as people confined in institutions.

Furthermore, the distribution of RS values for a given population can change significantly during the course of an epidemic, if vulnerable segments are subjected to additional health stressors, for example.

All-cause mortality analysis of COVID-19

In light of the above background and conceptual tools, we can now examine data for COVID-19, to date. For good reason (as per above), we ignore death-attributed data and model deconvolutions of P&I deaths versus other deaths deemed to be seasonal for reasons unrelated to the seasonal viral pathogens. We concentrate on all-cause mortality, by week.

All-cause mortality is not susceptible to bias, and is currently available for several jurisdictions. We use the raw data without any manipulation, and we do not modify the data to "correct" for changes in total population, or for changes in age structure of a population.

For the data, we rely on the CDC (USA), national institute data for England and Wales, and the graphical compilations of the EuroMOMO hub. We use only the latest weeks that are reported as complete (">100%", CDC) or reported to be of sufficient quality to publish. Unfortunately, some jurisdictions such as Canada can be characterized as slow and refractory to requests.

Figure 5 shows all-cause mortality by week for England and Wales, starting in 2010. The sudden single-week drops are book-keeping and death-certification-delay inconsistencies, which are counted in the following week(s). The red vertical line indicates the date at which the WHO declared the pandemic.

In declaring the pandemic, the WHO Director-General, Tedros Adhanom, put it this way, among other things:[2]

[2] "WHO Director-General's opening remarks at the media briefing on COVID-19 - 11 March 2020", https://www.who.int/dg/speeches/detail/who-director-general-s-opening-remarks-at-the-media-briefing-on-covid-19---11-march-2020

> […] In the days and weeks ahead, we expect to see the number of cases, the number of deaths, and the number of affected countries climb even higher. […]
> And we have called every day for countries to take urgent and aggressive action. We have rung the alarm bell loud and clear. […]
> This is not just a public health crisis, it is a crisis that will touch every sector – so every sector and every individual must be involved in the fight.
> I have said from the beginning that countries must take a whole-of-government, whole-of-society approach, built around a comprehensive strategy to prevent infections, save lives and minimize impact. […]
> I remind all countries that we are calling on you to activate and scale up your emergency response mechanisms; Communicate with your people about the risks and how they can protect themselves – this is everybody's business; Find, isolate, test and treat every case and trace every contact; Ready your hospitals; […] [my emphasis]

Adhanom's words either were the most remarkable public health forecast ever made for England and Wales (and many jurisdictions in the world, see below), or something else might explain the sharp peak in all-cause mortality that immediately followed his declaration.

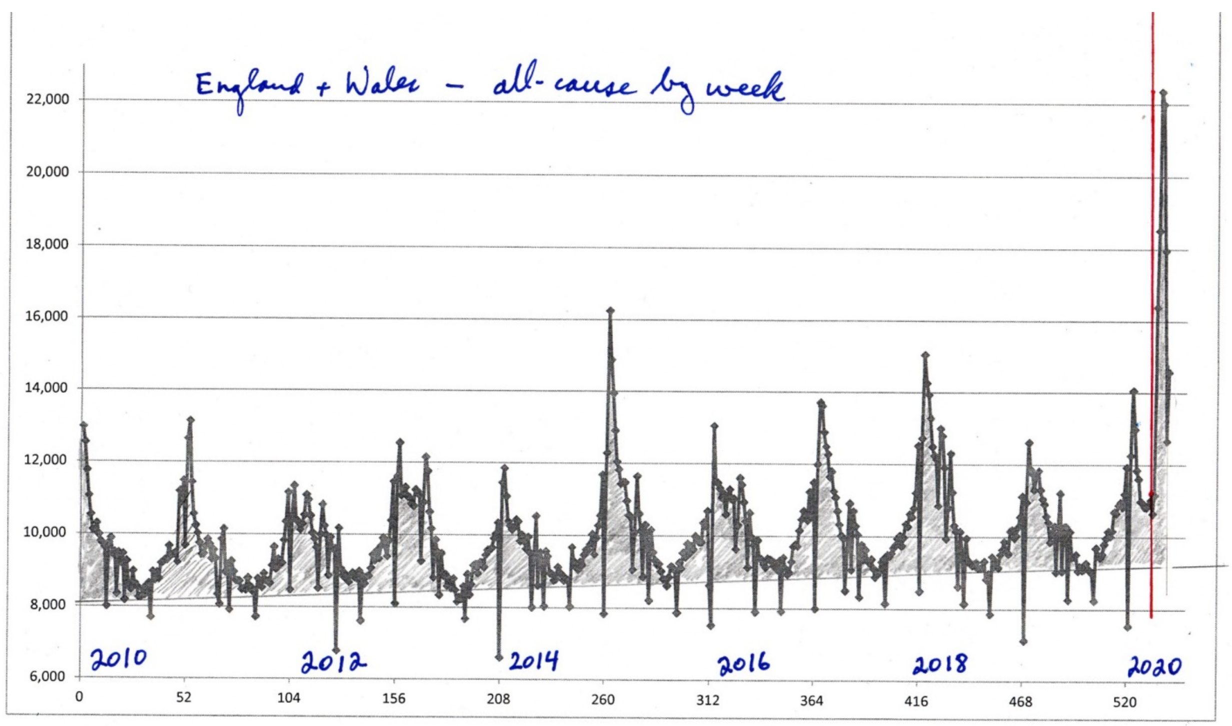


Figure 5: All-cause mortality by week for England and Wales, starting in 2010. The sudden single-week drops are book-keeping and death-certification-delay inconsistencies, which are counted in the following week(s). The red vertical line indicates the date at which the WHO declared the COVID-19 pandemic.

Importantly, the total number of winter-burden all-cause "excess" deaths for the season ending in 2020 (area above the summer baseline) is not statistically larger than for past years, and it remains to be seen how low the summer 2020 trough will be.

What can be called "the COVID peak" is a narrow feature (Figure 5). Relative to the summer baseline, the full-width at half-maximum of the peak is approximately 5 weeks. It has the distinction of being late in the infectious season, and of climbing far above the broader winter-burden hump.

This "COVID peak" is a unique event in the epidemiological history of England and Wales. Does this unique feature arise from an unusually novel viral pathogen, or does it arise from the unique, unprecedented and massive government response to the WHO declaration of a pandemic?

Note that such a "COVID peak" does not imply intrinsic virulence of the virus. It only means that the deaths of vulnerable persons, or persons made vulnerable, occurred in a short time span. For example, those who would have died in the next few or more weeks or months can have their deaths accelerated by human intervention, or those who are still recovering from a viral infection can be thrust into more precarious and stressful living conditions.

An analogous "COVID peak" occurred in the EuroMOMO hub data for Europe (Figure 6). Here again, the total number of winter-burden all-cause excess deaths for the season ending in 2020 (area above the summer baseline) is not statistically larger than for past years, and the date of declaration of the pandemic is shown by a vertical red line.

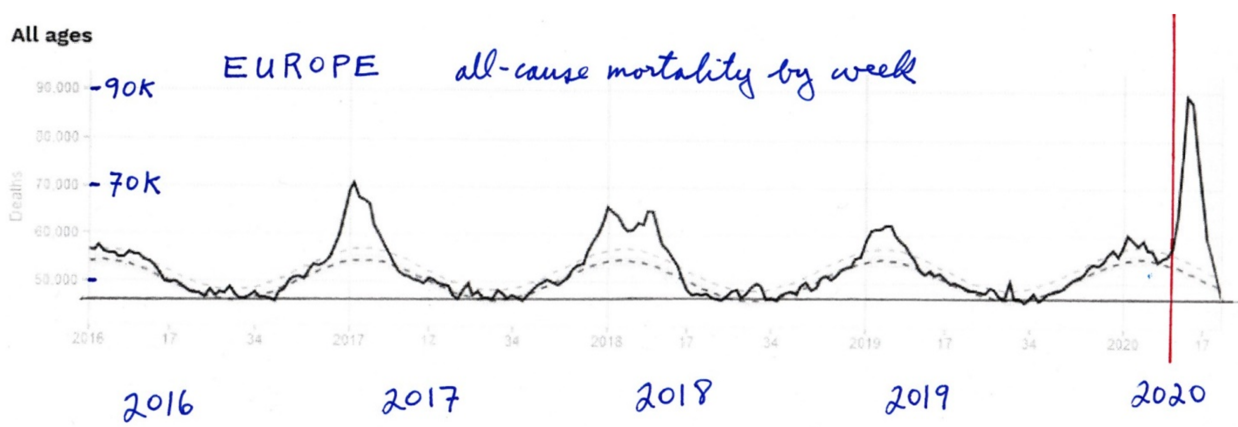


Figure 6: All-cause mortality by week EuroMOMO hub data for Europe, accessed on 1 June 2020. The date of declaration of the pandemic is shown by a vertical red line.

What looked like a concluding and "mild" 2020 season turned into a "COVID peak" immediately after the WHO declared the pandemic.

Let us next move to the USA, where both national and state-by-state current data is readily available, thanks to the CDC.

Figure 7 shows all-cause mortality by week for the USA, starting in 2014. Here the summer baseline is at approximately 46 K to 52 K deaths per week, increasing with the increase in total population. The red vertical line indicates the date at which the WHO declared the COVID-19 pandemic.

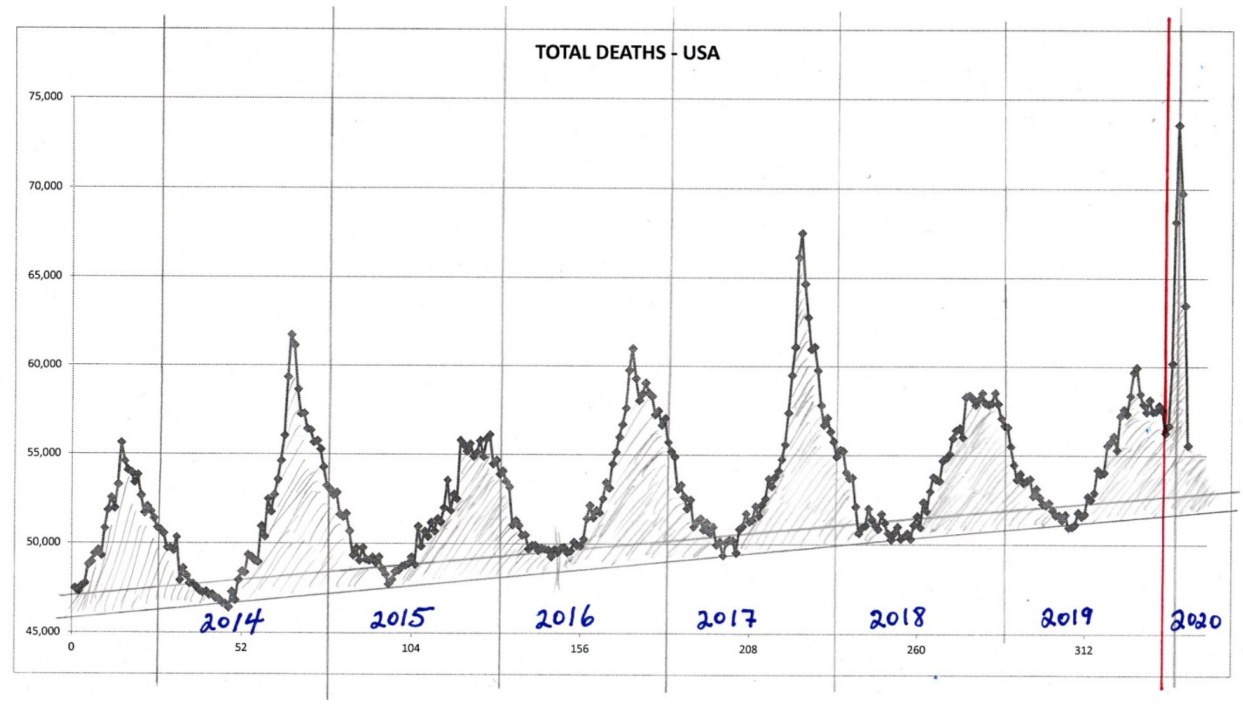


Figure 7: All-cause mortality by week for the USA, starting in 2014. The red vertical line indicates the date at which the WHO declared the COVID-19 pandemic. The hatched or gray-fill areas represent the all-cause winter-burden deaths for each year.

Here, again, we see that the total number of winter-burden all-cause deaths for the season ending in 2020 (area above the summer baseline) is not statistically larger than for past recent years. There is no evidence, purely in terms of number of seasonal deaths, to suggest any catastrophic event or exceptionally virulent pathogen. There was no “plague”. The winter burden, in these years, is consistently in the range of approximately 6% to 9% of total yearly all-cause mortality, and the year to year variations are typical of historic variations.

On the other hand, there is again a “COVID peak”, which has the following unique features:

- It is remarkably sharp or narrow, having a full-width at half-maximum of the peak, relative to the summer baseline, of approximately only 4 weeks. By comparison, the sharp peaks in the infectious seasons ending in 2015 and 2018 have such full-widths of 14 and 9 weeks, respectively.

- It occurs later in the infectious season than any other large sharp peak ever seen for the USA, surging after week-11 of 2020.

- Its surge occurs immediately after the WHO declared the pandemic, in perfect synchronicity, as seen in both Europe, and England and Wales, which are an ocean apart from the USA.

The “COVID peak” in the USA data arises from “hot spots”, such as New York City (NYC). Figure 8 shows the all-cause mortality by week for NYC, starting in 2013. The red vertical line indicates the date at which the WHO declared the COVID-19 pandemic.

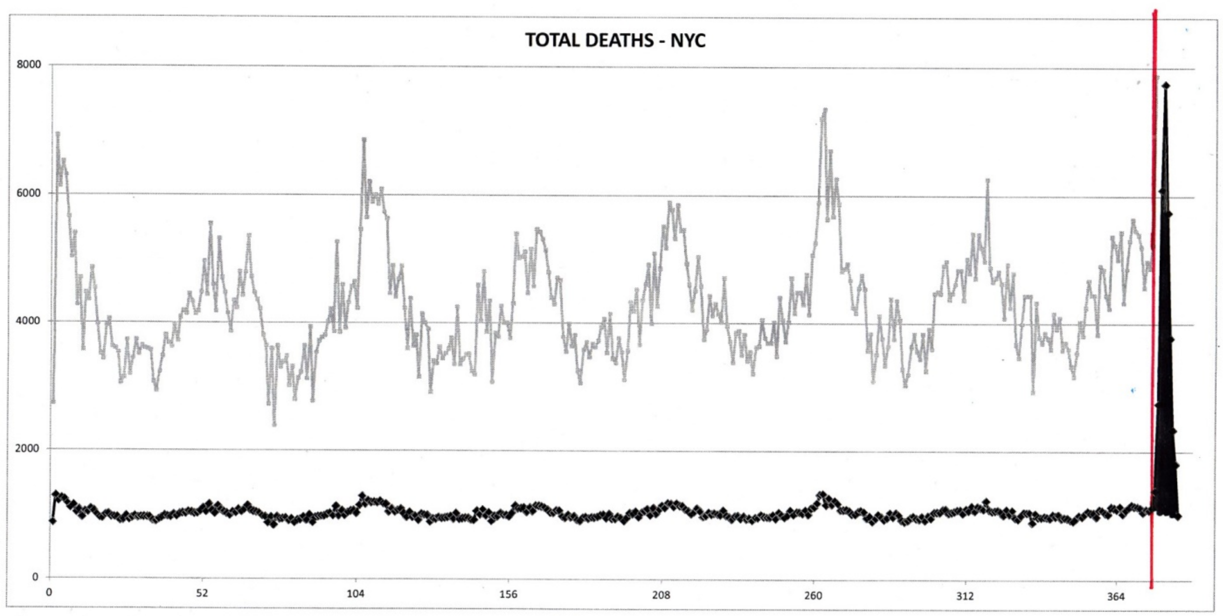


Figure 8: All-cause mortality by week for NYC, starting in 2013, in black. The red vertical line indicates the date at which the WHO declared the COVID-19 pandemic. The grey line is simply the same data on a vertically expanded and shifted scale, for visualization.

The NYC data makes no epidemiological sense whatsoever. The “COVID peak” here, on its face, cannot be interpreted as a normal viral respiratory disease process in a susceptible population. Local effects, such as importing patients from other jurisdictions or high densities of institutionalized or housed vulnerable people, must be in play, at least.

What is also striking is that some of the largest-population states in the USA, having large numbers of measured and reported cases, and large numbers of individuals with the antibodies, do not show a “COVID peak”. (Characteristic antibodies are produced and stored in the bodies of individuals who were infected and recovered following their immune responses. For example, see the antibody field study for California done by Bendavid et al., 2020).

This is shown for California in Figure 9, and for Texas in Figure 10.

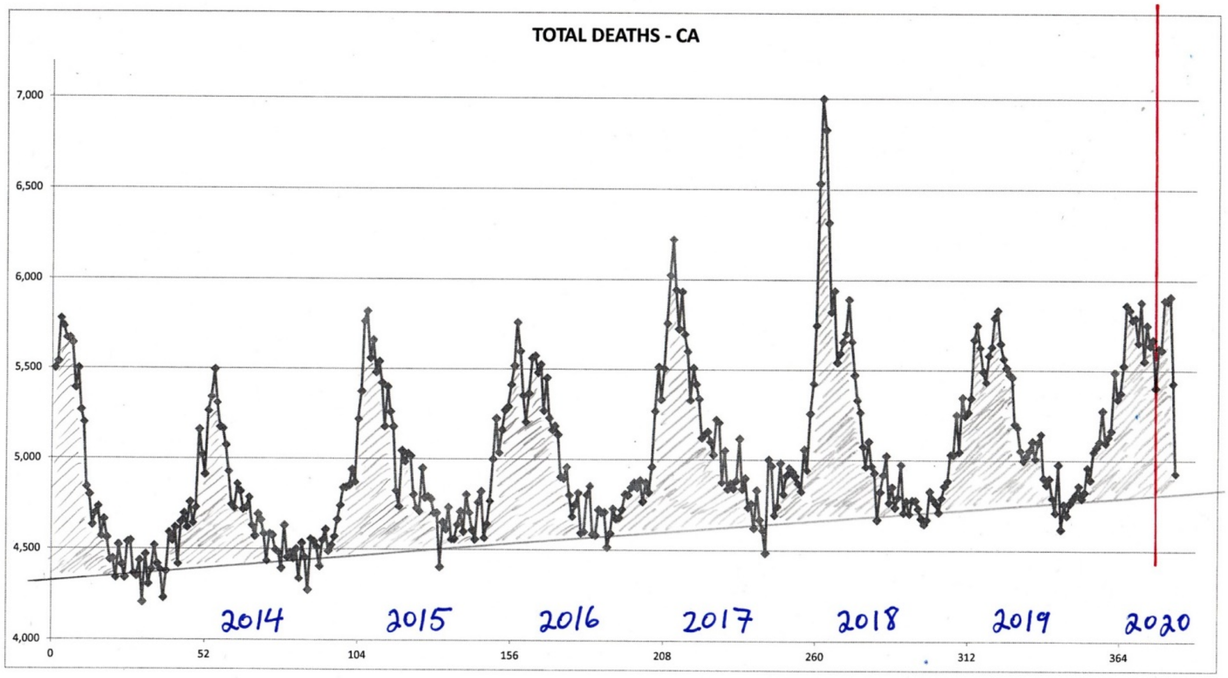


Figure 9: All-cause mortality by week for California, starting in 2013. The red vertical line indicates the date at which the WHO declared the COVID-19 pandemic. The hatched or gray-fill areas represent the all-cause winter-burden deaths for each year.

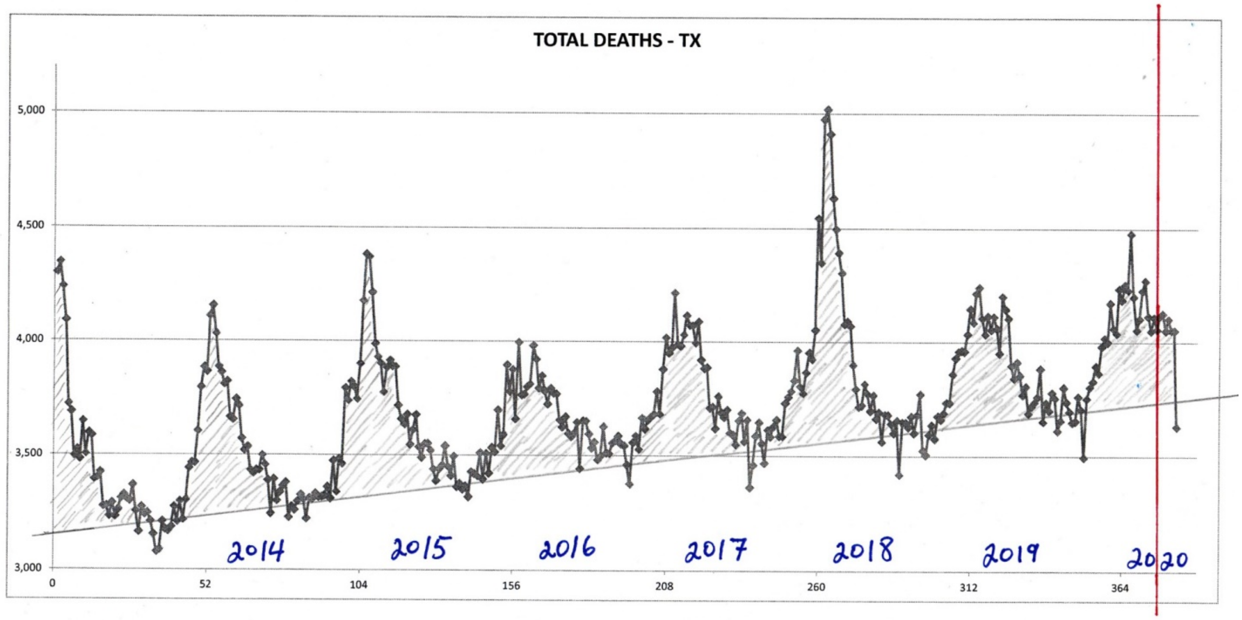
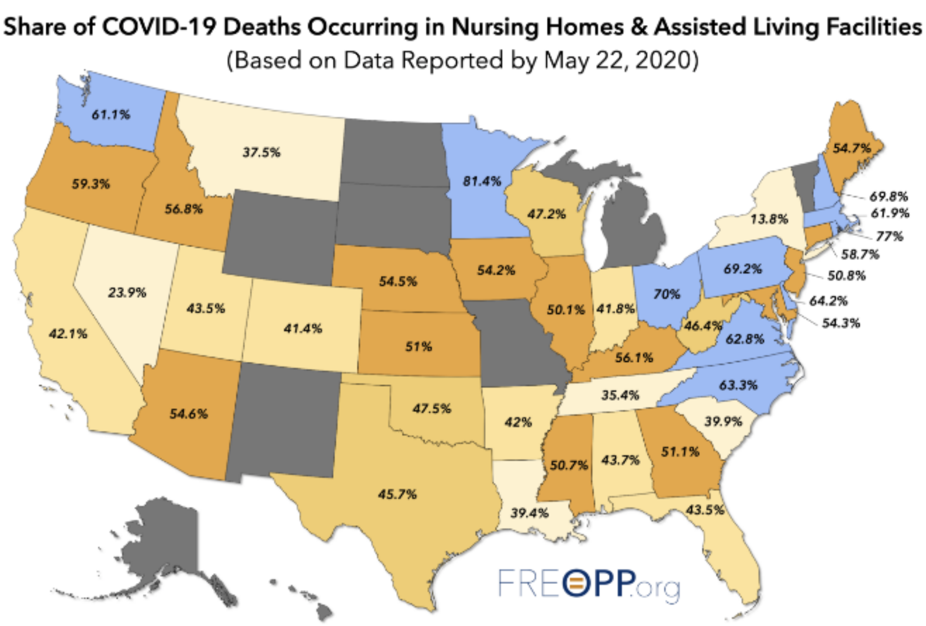


Figure 10: All-cause mortality by week for Texas, starting in 2013. The red vertical line indicates the date at which the WHO declared the COVID-19 pandemic. The hatched or gray-fill areas represent the all-cause winter-burden deaths for each year.

Also, none of the seven states that did not impose a lockdown (Iowa, Nebraska, North Dakota, South Dakota, Utah, Wyoming, and Arkansas) have a "COVID peak".

The presence of a "COVID peak" is positively correlated with the share of COVID-19-assigned deaths occurring in nursing homes and assisted living facilities, as per this map:



Interpreting the all-cause mortality "COVID peak"

Given the uniqueness of the all-cause mortality "COVID peak":

- Its sharpness, with a full-width at half-maximum of only approximately 4 weeks;
- Its lateness in the infectious-season cycle, surging after week-11 of 2020, which is unprecedented for any large sharp-peak feature;
- The synchronicity of the onset of its surge, across continents, and immediately following the WHO declaration of the pandemic; and
- Its USA state-to-state absence or presence for the same viral ecology on the same territory, being correlated with nursing home events and government actions rather than any known viral strain discernment.

Given the above review of knowledge about seasonal viral respiratory diseases:

- The robustly persistent and regular winter-burden patterns of all-cause mortality, across the modern era of epidemiology, and across nations in two hemispheres;
- The newfound (2010) understanding that transmissivity is controlled by absolute humidity, and that the transmission vector is small aerosol particles taken deeply into the lungs;
- The increasing recognition of metabolic energy budgeting as the paradigm for understanding death from infectious diseases with comorbidity conditions, while recognizing that the immune system has hierarchical control over metabolic energy budgeting, second only to cognition of external imminent danger; and
- The increasing understanding of the dominant role of metabolic stress (including stress cognition, perceived stress) in depressing immune system response capacity.

I postulate that the "COVID peak" represents an accelerated mass homicide of immune-vulnerable individuals, and individuals made more immune-vulnerable, by government and institutional actions, rather than being an epidemiological signature of a novel virus, irrespective of the degree to which the virus is novel from the perspective of viral speciation.

Finally, my interpretation of the "COVID peak" as being a signature of mass homicide by government response is supported by several institutional documents, media reports, and scientific articles, such as the following examples.

Two scientific articles are on-point:

- Hawryluck et al. (2004), on posttraumatic stress disorder (PTSD) arising from medical quarantine.

- Richardson et al. (2020), on statistical proof that mechanical ventilators killed critical COVID-19 patients.

Media articles and institutional memos include:

- "New study finds nearly all coronavirus patients put on ventilators died", *News Break | The Hill* 04-23, 23 April 2020.

https://www.newsbreak.com/news/0Oq9qI1z/new-study-finds-nearly-all-coronavirus-patients-put-on-ventilators-died

"New health care data suggests that almost half of all coronavirus patients placed on ventilators die, first reported by CNN. The data was gathered at Northwell Health, New York state's largest hospital system. It revealed that about 20 percent of COVID-19 patients passed away, and 88 percent of those placed on ventilators died."

- "Daughter blames 'chaos' of COVID-19 pandemic for mother's rapid decline", by Arthur White-Crummey, *Regina Leader-Post*, 29 May 2020.

https://thestarphoenix.com/news/saskatchewan/daughter-blames-chaos-of-covid-19-pandemic-for-mothers-rapid-decline/

"Sue Nimegeers's mother never had COVID-19, but she still counts her as a victim of the disease. "She never tested positive, but the chaos of the pandemic itself around us, we feel, took her from us just way too soon," Nimegeers told the board of the Saskatchewan Health Authority (SHA) on Friday."

- " 'Deeply disturbing' report into Ontario care homes released", *BBC*, 27 May 2020.

https://www.bbc.com/news/world-us-canada-52814435

"Mr Ford said a full investigation has been launched into the allegations, which included claims that facilities smelt of rotten food, infested with cockroaches and flies, and that elderly people were left for hours "crying for help with staff not responding"."

- "Nothing can justify this destruction of people's lives", Yoram Lass, former director of Israel's Health Ministry, on the hysteria around Covid-19, *sp!ked*, 22 May 2020.

https://www.spiked-online.com/2020/05/22/nothing-can-justify-this-destruction-of-peoples-lives/

"Yoram Lass: It is the first epidemic in history which is accompanied by another epidemic – the virus of the social networks. These new media have brainwashed entire populations. What you get is fear and anxiety, and an inability to look at real data. And therefore you have all the ingredients for monstrous hysteria.
It is what is known in science as positive feedback or a snowball effect. The government is afraid of its constituents. Therefore, it implements draconian measures. The constituents look at the draconian measures and become even more hysterical."

- “Cuomo downplays calls for federal probe into nursing home coronavirus deaths: 'Ask President Trump' “, by Andrew O'Reilly | *Fox News*, 20 May 2020.

https://www.foxnews.com/politics/cuomo-probe-into-nursing-home-coronavirus-deaths-ask-president-trump

“New York Gov. Andrew Cuomo on Wednesday brushed off calls for the Department of Justice to open an investigation into the massive number of deaths in the state’s nursing homes during the coronavirus pandemic – claiming he was only following guidelines from the Trump administration and Centers for Disease Control and Prevention.
While no formal probe has been announced, the speculation comes amid scrutiny of his March 25 directive that required nursing homes to take on new patients infected with COVID-19.”

- DATE: March 25, 2020

TO: Nursing Home Administrators, Directors of Nursing, and Hospital Discharge Planners
FROM: New York State Department of Health
Advisory: Hospital Discharges and Admissions to Nursing Homes
(Removed from:
https://coronavirus.health.ny.gov/system/files/documents/2020/03/doh_covid19-_nhadmissionsreadmissions_-032520.pdf)

“During this global health emergency, all NHs must comply with the expedited receipt of residents returning from hospitals to NHs. Residents are deemed appropriate for return to a NH upon a determination by the hospital physician or designee that the resident is medically stable for return. […]
No resident shall be denied re-admission or admission to the NH solely based on a confmned or suspected diagnosis ofCOVID-19. NHs are prohibited from requiring a hospitalized resident who is determined medically stable to be tested for COVID-19 prior to admission or readmission.”

- “Nursing Homes & Assisted Living Facilities Account for 42% of COVID-19 Deaths: A startling statistic has profound implications for the way we’ve managed the coronavirus pandemic”, by Gregg Girvan, *FREOPP*, 7 May 2020.

https://freopp.org/the-covid-19-nursing-home-crisis-by-the-numbers-3a47433c3f70

“Based on a new analysis of state-by-state COVID-19 fatality reports, it is clear that the most underappreciated aspect of the novel coronavirus pandemic is its effect on a specific population of Americans: those living in nursing homes and assisted living facilities.”

- “Guilty - Of Breathing”, by Tony Heller, *Tony Heller YouTube Channel*, 24 May 2020.

https://www.youtube.com/watch?v=4sjNQ4YTUM4

“Lockdowns were sold months ago on the idea of "flattening the curve." In most places there never was much of a curve to flatten, yet the lockdowns are still in place. Tens of millions are now having their lives destroyed - for the crime of breathing.”

- “The 'massacre' of Italy's elderly nursing home residents: Covid-19 patients in Italy's virus epicentre of Lombardy were transferred to nursing homes by an official resolution with catastrophic consequences”, by Maria Tavernini and Alessandro Di Rienzo, *TRT World*, 20 April 2020.

https://www.trtworld.com/magazine/the-massacre-of-italy-s-elderly-nursing-home-residents-35575

“Hosting Covid-19 patients in nursing homes was like lighting a match in a haystack.”

- “Coronavirus Update: How shoring up hospitals for COVID-19 contributed to Canada’s long-term care crisis”, by Jessie Willms and Hailey Montgomery, *Globe & Mail*, 20 May 2020.

https://www.theglobeandmail.com/canada/article-coronavirus-update-how-shoring-up-hospitals-for-covid-19-contributed/

“Most of the nursing- and retirement-home residents who have succumbed to COVID-19 in Canada died inside the virus-stricken, understaffed facilities as hospital beds sat empty.”

- “There Is No Evidence Lockdowns Saved Lives. It Is Indisputable They Caused Great Harm”, by Briggs, *wmbriggs.com*, 14 May 2020.

https://wmbriggs.com/post/30833/

“In the end, it does not come down to country- or even city-level statistics. It comes down to people. Each individual catches the bug or not, lives or dies. Not because of their country, but because of themselves, their health, their circumstances. Any given individual might have benefited from self-quarantine and loss of job. Just as any given individual might have come to a bad end from a lockdown.”

- “Hospitals get paid more to list patients as COVID-19”, by Tom Kertscher, *POLITIFACT*, 21 April 2020.

https://www.politifact.com/factchecks/2020/apr/21/facebook-posts/Fact-check-Hospitals-COVID-19-payments/

“It’s standard for Medicare to pay a hospital roughly three times as much for a patient who goes on a ventilator, as for one who doesn’t. Medicare is paying a 20% add-on to its regular hospital payments for the treatment of COVID-19 victims. That’s a result of a federal stimulus law.”

- “CDC: 80,000 people died of flu last winter in U.S., highest death toll in 40 years”, by Associated Press, *STAT News*, 26 September 2018.

https://www.statnews.com/2018/09/26/cdc-us-flu-deaths-winter/

“An estimated 80,000 Americans died of flu and its complications last winter — the disease’s highest death toll in at least four decades. The director of the Centers for Disease Control and Prevention, Dr. Robert Redfield, revealed the total in an interview Tuesday night with The Associated Press.”

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Masks Don't Work: A review of science relevant to COVID-19 social policy

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D. G. Rancourt
Ontario Civil Liberties Association

Masks Don't Work

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Denis G. Rancourt, PhD
Researcher, Ontario Civil Liberties Association (ocla.ca)

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Summary / Abstract

Masks and respirators do not work.

There have been extensive randomized controlled trial (RCT) studies, and meta-analysis reviews of RCT studies, which all show that masks and respirators do not work to prevent respiratory influenza-like illnesses, or respiratory illnesses believed to be transmitted by droplets and aerosol particles.

Furthermore, the relevant known physics and biology, which I review, are such that masks and respirators should not work. It would be a paradox if masks and respirators worked, given what we know about viral respiratory diseases: The main transmission path is long-residence-time aerosol particles (< 2.5 μm), which are too fine to be blocked, and the minimum-infective-dose is smaller than one aerosol particle.

The present paper about masks illustrates the degree to which governments, the mainstream media, and institutional propagandists can decide to operate in a science vacuum, or select only incomplete science that serves their interests. Such recklessness is also certainly the case with the current global lockdown of over 1 billion people, an unprecedented experiment in medical and political history.

Review of the Medical Literature

Here are key anchor points to the extensive scientific literature that establishes that wearing surgical masks and respirators (e.g., "N95") does not reduce the risk of contracting a verified illness:

> **Jacobs, J. L. et al. (2009)** "Use of surgical face masks to reduce the incidence of the common cold among health care workers in Japan: A randomized controlled trial", *American Journal of Infection Control*, Volume 37, Issue 5, 417 - 419.
> https://www.ncbi.nlm.nih.gov/pubmed/19216002
>
>> N95-masked health-care workers (HCW) were significantly more likely to experience headaches. Face mask use in HCW was not demonstrated to provide benefit in terms of cold symptoms or getting colds.

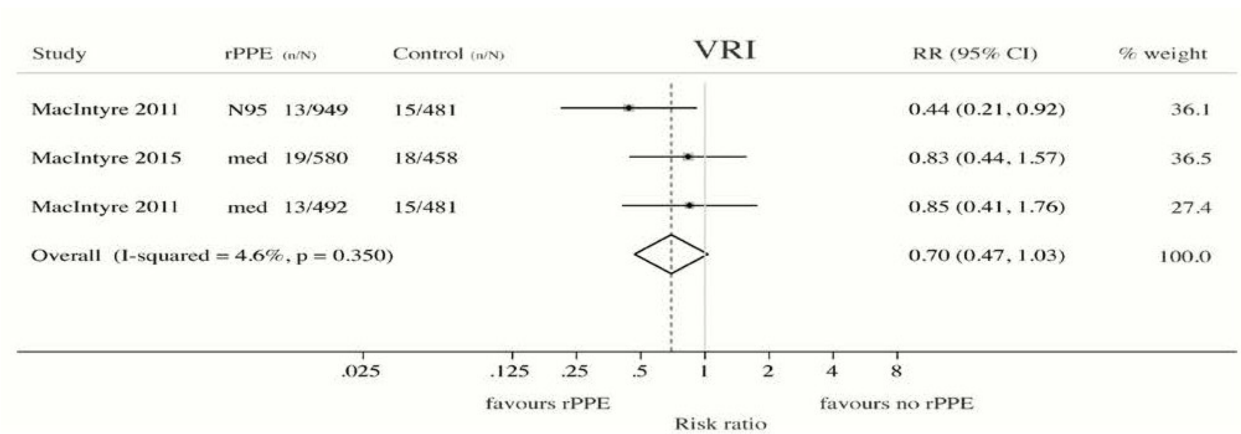
> **Cowling, B. et al. (2010)** "Face masks to prevent transmission of influenza virus: A systematic review", *Epidemiology and Infection*, 138(4), 449-456. doi:10.1017/S0950268809991658
> https://www.cambridge.org/core/journals/epidemiology-and-infection/article/face-masks-to-prevent-transmission-of-influenza-virus-a-systematic-review/64D368496EBDE0AFCC6639CCC9D8BC05
>
>> None of the studies reviewed showed a benefit from wearing a mask, in either HCW or community members in households (H). See summary Tables 1 and 2 therein.

> **bin-Reza et al. (2012)** "The use of masks and respirators to prevent transmission of influenza: a systematic review of the scientific evidence", *Influenza and Other Respiratory Viruses* 6(4), 257–267.
> https://onlinelibrary.wiley.com/doi/epdf/10.1111/j.1750-2659.2011.00307.x
>
>> "There were 17 eligible studies. … None of the studies established a conclusive relationship between mask ⁄ respirator use and protection against influenza infection."

> **Smith, J.D. et al. (2016)** "Effectiveness of N95 respirators versus surgical masks in protecting health care workers from acute respiratory infection: a systematic review and meta-analysis", *CMAJ* Mar 2016, cmaj.150835; DOI: 10.1503/cmaj.150835
> https://www.cmaj.ca/content/188/8/567
>
>> "We identified 6 clinical studies ... In the meta-analysis of the clinical studies, we found no significant difference between N95 respirators and surgical masks in associated risk of (a) laboratory-confirmed respiratory infection, (b) influenza-like illness, or (c) reported work-place absenteeism."

Offeddu, V. et al. (2017) "Effectiveness of Masks and Respirators Against Respiratory Infections in Healthcare Workers: A Systematic Review and Meta-Analysis", *Clinical Infectious Diseases*, Volume 65, Issue 11, 1 December 2017, Pages 1934–1942, https://doi.org/10.1093/cid/cix681
https://academic.oup.com/cid/article/65/11/1934/4068747

> "Self-reported assessment of clinical outcomes was prone to bias. Evidence of a protective effect of masks or respirators against verified respiratory infection (VRI) was not statistically significant"; as per Fig. 2c therein:



Radonovich, L.J. et al. (2019) "N95 Respirators vs Medical Masks for Preventing Influenza Among Health Care Personnel: A Randomized Clinical Trial", *JAMA*. 2019; 322(9): 824–833. doi:10.1001/jama.2019.11645
https://jamanetwork.com/journals/jama/fullarticle/2749214

> "Among 2862 randomized participants, 2371 completed the study and accounted for 5180 HCW-seasons. … Among outpatient health care personnel, N95 respirators vs medical masks as worn by participants in this trial resulted in no significant difference in the incidence of laboratory-confirmed influenza."

Long, Y. et al. (2020) "Effectiveness of N95 respirators versus surgical masks against influenza: A systematic review and meta-analysis", *J Evid Based Med.* 2020; 1- 9. https://doi.org/10.1111/jebm.12381
https://onlinelibrary.wiley.com/doi/epdf/10.1111/jebm.12381

> "A total of six RCTs involving 9 171 participants were included. There were no statistically significant differences in preventing laboratory-confirmed influenza, laboratory-confirmed respiratory viral infections, laboratory-confirmed respiratory infection and influenza-like illness using N95 respirators and surgical masks. Meta-analysis indicated a protective effect of N95 respirators against laboratory-confirmed bacterial colonization (RR = 0.58, 95% CI 0.43-0.78). The

use of N95 respirators compared with surgical masks is not associated with a lower risk of laboratory-confirmed influenza."

Conclusion Regarding that Masks Do Not Work

No RCT study with verified outcome shows a benefit for HCW or community members in households to wearing a mask or respirator. There is no such study. There are no exceptions.

Likewise, no study exists that shows a benefit from a broad policy to wear masks in public (more on this below).

Furthermore, if there were any benefit to wearing a mask, because of the blocking power against droplets and aerosol particles, then there should be more benefit from wearing a respirator (N95) compared to a surgical mask, yet several large meta-analyses, and all the RCT, prove that there is no such relative benefit.

Masks and respirators do not work.

Precautionary Principle Turned on Its Head with Masks

In light of the medical research, therefore, it is difficult to understand why public-health authorities are not consistently adamant about this established scientific result, since the distributed psychological, economic and environmental harm from a broad recommendation to wear masks is significant, not to mention the unknown potential harm from concentration and distribution of pathogens on and from used masks. In this case, public authorities would be turning the precautionary principle on its head (see below).

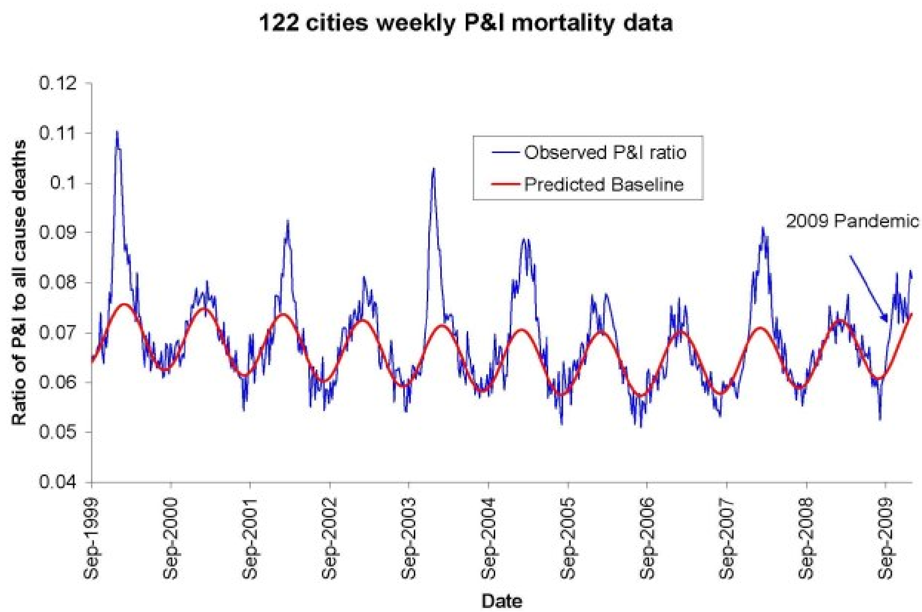
Physics and Biology of Viral Respiratory Disease and of Why Masks Do Not Work

In order to understand why masks cannot possibly work, we must review established knowledge about viral respiratory diseases, the mechanism of seasonal variation of excess deaths from pneumonia and influenza, the aerosol mechanism of infectious disease transmission, the physics and chemistry of aerosols, and the mechanism of the so-called minimum-infective-dose.

In addition to pandemics that can occur anytime, in the temperate latitudes there is an extra burden of respiratory-disease mortality that is seasonal, and that is caused by viruses. For

example, see the review of influenza by Paules and Subbarao (2017). This has been known for a long time, and the seasonal pattern is exceedingly regular.

For example, see Figure 1 of Viboud (2010), which has “Weekly time series of the ratio of deaths from pneumonia and influenza to all deaths, based on the 122 cities surveillance in the US (blue line). The red line represents the expected baseline ratio in the absence of influenza activity,” here:



The seasonality of the phenomenon was largely not understood until a decade ago. Until recently, it was debated whether the pattern arose primarily because of seasonal change in virulence of the pathogens, or because of seasonal change in susceptibility of the host (such as from dry air causing tissue irritation, or diminished daylight causing vitamin deficiency or hormonal stress). For example, see Dowell (2001).

In a landmark study, Shaman et al. (2010) showed that the seasonal pattern of extra respiratory-disease mortality can be explained quantitatively on the sole basis of absolute humidity, and its direct controlling impact on transmission of airborne pathogens.

Lowen et al. (2007) demonstrated the phenomenon of humidity-dependent airborne-virus virulence in actual disease transmission between guinea pigs, and discussed potential underlying mechanisms for the measured controlling effect of humidity.

The underlying mechanism is that the pathogen-laden aerosol particles or droplets are neutralized within a half-life that monotonically and significantly decreases with increasing ambient humidity. This is based on the seminal work of Harper (1961). Harper experimentally showed that viral-pathogen-carrying droplets were inactivated within shorter and shorter times, as ambient humidity was increased.

Harper argued that the viruses themselves were made inoperative by the humidity ("viable decay"), however, he admitted that the effect could be from humidity-enhanced physical removal or sedimentation of the droplets ("physical loss"): "Aerosol viabilities reported in this paper are based on the ratio of virus titre to radioactive count in suspension and cloud samples, and can be criticized on the ground that test and tracer materials were not physically identical."

The latter ("physical loss") seems more plausible to me, since humidity would have a universal physical effect of causing particle / droplet growth and sedimentation, and all tested viral pathogens have essentially the same humidity-driven "decay". Furthermore, it is difficult to understand how a virion (of all virus types) in a droplet would be molecularly or structurally attacked or damaged by an increase in ambient humidity. A "virion" is the complete, infective form of a virus outside a host cell, with a core of RNA or DNA and a capsid. The actual mechanism of such humidity-driven intra-droplet "viable decay" of a virion has not been explained or studied.

In any case, the explanation and model of Shaman et al. (2010) is not dependant on the particular mechanism of the humidity-driven decay of virions in aerosol / droplets. Shaman's quantitatively demonstrated model of seasonal regional viral epidemiology is valid for either mechanism (or combination of mechanisms), whether "viable decay" or "physical loss".

The breakthrough achieved by Shaman et al. is not merely some academic point. Rather, it has profound health-policy implications, which have been entirely ignored or overlooked in the current coronavirus pandemic.

In particular, Shaman's work necessarily implies that, rather than being a fixed number (dependent solely on the spatial-temporal structure of social interactions in a completely susceptible population, and on the viral strain), the epidemic's **basic reproduction number** (R0) is highly or predominantly dependent on ambient absolute humidity.

For a definition of R0, see HealthKnowlege-UK (2020): R0 is "the average number of secondary infections produced by a typical case of an infection in a population where everyone is susceptible." The average R0 for influenza is said to be 1.28 (1.19–1.37); see the comprehensive review by Biggerstaff et al. (2014).

In fact, Shaman et al. showed that R0 must be understood to seasonally vary between humid-summer values of just larger than "1" and dry-winter values typically as large as "4" (for example, see their Table 2). In other words, the seasonal infectious viral respiratory diseases that plague temperate latitudes every year go from being intrinsically mildly contagious to

virulently contagious, due simply to the bio-physical mode of transmission controlled by atmospheric humidity, irrespective of any other consideration.

Therefore, all the epidemiological mathematical modelling of the benefits of mediating policies (such as social distancing), which assumes humidity-independent R0 values, has a large likelihood of being of little value, on this basis alone. For studies about modelling and regarding mediation effects on the effective reproduction number, see Coburn (2009) and Tracht (2010).

To put it simply, the "second wave" of an epidemic is not a consequence of human sin regarding mask wearing and hand shaking. Rather, the "second wave" is an inescapable consequence of an air-dryness-driven many-fold increase in disease contagiousness, in a population that has not yet attained immunity.

If my view of the mechanism is correct (i.e., "physical loss"), then Shaman's work further necessarily implies that the dryness-driven high transmissibility (large R0) arises from small aerosol particles fluidly suspended in the air; as opposed to large droplets that are quickly gravitationally removed from the air.

Such small aerosol particles fluidly suspended in air, of biological origin, are of every variety and are everywhere, including down to virion-sizes (Despres, 2012). It is not entirely unlikely that viruses can thereby be physically transported over inter-continental distances (e.g., Hammond, 1989).

More to the point, indoor airborne virus concentrations have been shown to exist (in day-care facilities, health centres, and onboard airplanes) primarily as aerosol particles of diameters smaller than 2.5 µm, such as in the work of Yang et al. (2011):

> "Half of the 16 samples were positive, and their total virus concentrations ranged from 5800 to 37 000 genome copies m^{-3}. On average, 64 per cent of the viral genome copies were associated with fine particles smaller than 2.5 µm, which can remain suspended for hours. Modelling of virus concentrations indoors suggested a source strength of $1.6 \pm 1.2 \times 10^5$ genome copies m^{-3} air h^{-1} and a deposition flux onto surfaces of 13 ± 7 genome copies m^{-2} h^{-1} by Brownian motion. Over 1 hour, the inhalation dose was estimated to be 30 ± 18 median tissue culture infectious dose ($TCID_{50}$), adequate to induce infection. These results provide quantitative support for the idea that the aerosol route could be an important mode of influenza transmission."

Such small particles (< 2.5 µm) are part of air fluidity, are not subject to gravitational sedimentation, and would not be stopped by long-range inertial impact. This means that the slightest (even momentary) facial misfit of a mask or respirator renders the design filtration norm of the mask or respirator entirely irrelevant. In any case, the filtration material itself of

N95 (average pore size ~0.3−0.5 µm) does not block virion penetration, not to mention surgical masks. For example, see Balazy et al. (2006).

Mask stoppage efficiency and host inhalation are only half of the equation, however, because the minimal infective dose (MID) must also be considered. For example, if a large number of pathogen-laden particles must be delivered to the lung within a certain time for the illness to take hold, then partial blocking by any mask or cloth can be enough to make a significant difference.

On the other hand, if the MID is amply surpassed by the virions carried in a single aerosol particle able to evade mask-capture, then the mask is of no practical utility, which is the case.

Yezli and Otter (2011), in their review of the MID, point out relevant features:

- most respiratory viruses are as infective in humans as in tissue culture having optimal laboratory susceptibility
- it is believed that a single virion can be enough to induce illness in the host
- the 50%-probability MID ("$TCID_{50}$") has variably been found to be in the range 100−1000 virions
- there are typically 10^3−10^7 virions per aerolized influenza droplet with diameter 1 µm − 10 µm
- the 50%-probability MID easily fits into a single (one) aerolized droplet

For further background:

- A classic description of dose-response assessment is provided by Haas (1993).
- Zwart et al. (2009) provided the first laboratory proof, in a virus-insect system, that the action of a single virion can be sufficient to cause disease.
- Baccam et al. (2006) calculated from empirical data that, with influenza A in humans, "we estimate that after a delay of ~6 h, infected cells begin producing influenza virus and continue to do so for ~5 h. The average lifetime of infected cells is ~11 h, and the half-life of free infectious virus is ~3 h. We calculated the [in-body] basic reproductive number, R_0, which indicated that a single infected cell could produce ~22 new productive infections."
- Brooke et al. (2013) showed that, contrary to prior modeling assumptions, although not all influenza-A-infected cells in the human body produce infectious progeny (virions), nonetheless, 90% of infected cell are significantly impacted, rather than simply surviving unharmed.

All of this to say that: if anything gets through (and it always does, irrespective of the mask), then you are going to be infected. Masks cannot possibly work. It is not surprising, therefore, that no bias-free study has ever found a benefit from wearing a mask or respirator in this application.

Therefore, the studies that show partial stopping power of masks, or that show that masks can capture many large droplets produced by a sneezing or coughing mask-wearer, in light of the above-described features of the problem, are irrelevant. For example, such studies as these: Leung (2020), Davies (2013), Lai (2012), and Sande (2008).

Why There Can Never Be an Empirical Test of a Nation-Wide Mask-Wearing Policy

As mentioned above, no study exists that shows a benefit from a broad policy to wear masks in public. There is good reason for this. It would be impossible to obtain unambiguous and bias-free results:

- Any benefit from mask-wearing would have to be a small effect, since undetected in controlled experiments, which would be swamped by the larger effects, notably the large effect from changing atmospheric humidity.
- Mask compliance and mask adjustment habits would be unknown.
- Mask-wearing is associated (correlated) with several other health behaviours; see Wada (2012).
- The results would not be transferable, because of differing cultural habits.
- Compliance is achieved by fear, and individuals can habituate to fear-based propaganda, and can have disparate basic responses.
- Monitoring and compliance measurement are near-impossible, and subject to large errors.
- Self-reporting (such as in surveys) is notoriously biased, because individuals have the self-interested belief that their efforts are useful.
- Progression of the epidemic is not verified with reliable tests on large population samples, and generally relies on non-representative hospital visits or admissions.
- Several different pathogens (viruses and strains of viruses) causing respiratory illness generally act together, in the same population and/or in individuals, and are not resolved, while having different epidemiological characteristics.

Unknown Aspects of Mask Wearing

Many potential harms may arise from broad public policies to wear masks, and the following unanswered questions arise:

- Do used and loaded masks become sources of enhanced transmission, for the wearer and others?

- Do masks become collectors and retainers of pathogens that the mask wearer would otherwise avoid when breathing without a mask?
- Are large droplets captured by a mask atomized or aerolized into breathable components? Can virions escape an evaporating droplet stuck to a mask fiber?
- What are the dangers of bacterial growth on a used and loaded mask?
- How do pathogen-laden droplets interact with environmental dust and aerosols captured on the mask?
- What are long-term health effects on HCW, such as headaches, arising from impeded breathing?
- Are there negative social consequences to a masked society?
- Are there negative psychological consequences to wearing a mask, as a fear-based behavioural modification?
- What are the environmental consequences of mask manufacturing and disposal?
- Do the masks shed fibres or substances that are harmful when inhaled?

Conclusion

By making mask-wearing recommendations and policies for the general public, or by expressly condoning the practice, governments have both ignored the scientific evidence and done the opposite of following the precautionary principle.

In an absence of knowledge, governments should not make policies that have a hypothetical potential to cause harm. The government has an onus barrier before it instigates a broad social-engineering intervention, or allows corporations to exploit fear-based sentiments.

Furthermore, individuals should know that there is no known benefit arising from wearing a mask in a viral respiratory illness epidemic, and that scientific studies have shown that any benefit must be residually small, compared to other and determinative factors.

Otherwise, what is the point of publicly funded science?

The present paper about masks illustrates the degree to which governments, the mainstream media, and institutional propagandists can decide to operate in a science vacuum, or select only incomplete science that serves their interests. Such recklessness is also certainly the case with the current global lockdown of over 1 billion people, an unprecedented experiment in medical and political history.

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GEO-ECONOMICS AND GEO-POLITICS DRIVE SUCCESSIVE ERAS OF PREDATORY GLOBALIZATION AND SOCIAL ENGINEERING

Historical emergence of climate change, gender equity, and anti-racism as State doctrines

Denis G. Rancourt, PhD
Researcher

Ontario Civil Liberties Association
603-170 Laurier Avenue West
Ottawa, Ontario
Canada K1P 5V5
http://ocla.ca

Geo-Economics and Geo-Politics Drive Successive Eras of Predatory Globalization and Social Engineering[1]

Historical emergence of climate change, gender equity, and anti-racism as State doctrines

Denis G. Rancourt, PhD
Researcher
Ontario Civil Liberties Association (ocla.ca)



Introduction / Abstract

The influence of geopolitical and global economic conditions on the fabric of domestic societies and on individual psychology is most frequently underestimated by civilian commentators, especially regarding Western "free and democratic" societies. The military, on the other hand, do not underestimate the importance of broad trade and economic factors on the very fabric of a society and on the psychology of its citizens, at least in targeted developing countries.[2]

This article has two main goals.

The first is to demonstrate the large extent to which the global financial system determines national and regional reality in people's lives and security, including in the USA itself and in the Western world in general, with an emphasis on the two main post-World-War-II transformations, which were initiated in 1971, following the cancellation of the Bretton Woods agreement, and in 1991, following the dissolution of the Soviet Union.

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[1] Cite this report as: *"Geo-Economics and Geo-Politics Drive Successive Eras of Predatory Globalization and Social Engineering: Historical emergence of climate change, gender equity, and anti-racism as State doctrines", by Denis G. Rancourt, Ontario Civil Liberties Association, OCLA Report 2019-1, April 2019,* http://ocla.ca/OCLA_Report_2019-1/.

[2] So-called sanctions (and trade blockades) and currency devaluation are weapons of mass destruction, often applied prior to economic restructuring or military obliteration, just as "winning hearts and minds" is a weapon of economic occupation. And see: "Leaked Wikileaks Doc Reveals US Military Use of IMF, World Bank as "Unconventional" Weapons — This "U.S. coup manual," recently highlighted by WikiLeaks, serves as a reminder that the so-called "independence" of such financial institutions as The World Bank and IMF is an illusion and that they are among the many "financial weapons" regularly used by the U.S. government to bend countries to its will." By Whitney Webb, *MintPress News*, 7 February 2019, https://www.mintpressnews.com/leaked-wikileaks-doc-reveals-how-us-military-uses-of-imf-world-bank-as-unconventional-weapons/254708/.

The second is to describe the on-going tectonic shift that followed the 1991 dissolution of the Soviet Union in broader terms than is usually envisioned, and how this driven and coordinated shift was chronologically accompanied by: a dramatic acceleration of trade and finance “globalization”, and an unprecedented campaign of social engineering of the Western upper-middle-classes, aimed at facilitating USA and world-elite opportunistic exploitation of the new global circumstances, in turn leading to the present Gilets jaunes, Brexit, Trump… backlash. (In a sense, “the Russians did it.”)

For an “executive summary” with description of the supporting socio-economic data, see the Conclusion section.

TABLE OF CONTENTS

PART-I: ECONOMIC GLOBALIZATION

Bretton Woods

The deciding architects of the post-Second-World-War (post-WWII) Bretton Woods system of global finance wanted stable conditions for the growth and reconstruction of the USA-led "free world", to avoid wars and destabilizing economic maneuvering between capitalist Western states, and to facilitate military and strategic integration under USA supremacy, while allowing "fair" exploitation of the "developing world" and Western allies, under USA control.

The resulting Bretton Woods system had three operational components [1][2]:

> 1. "[T]he American dollar functioned as a virtual world currency, conferring great advantages on the US vis-à-vis the other capitalist powers. These advantages were limited, at least in theory, by the provision that the US dollar could be redeemed in gold at the rate of $35 per ounce."[2]
>
> 2. There were mechanisms to ensure balance of trade accounts, including the control of currency exchange rates. No signatory nation could accumulate an excessive deficit or surplus.

> 3. Capital mobility (flight) was limited, in order to preserve some national economic sovereignty: “Keynes had made clear that if free capital movements were allowed then it would not be possible to establish the kind of regulated capitalism at which the new agreement was aimed. ‘Freedom of capital movements,’ he insisted, ‘is an essential part of the old laissez-faire system and assumes that it is right to have an equalisation of interest rates in all parts of the world. ... In my view the whole management of the domestic economy depends upon being free to have the appropriate rate of interest without reference to the rates prevailing elsewhere in the world. Capital control is a corollary to this.’”[2]

Obviously, unilateral sanctions against signatories (against members of the “free world” and its protectorates) were not possible.

A foreseen flaw with Bretton Woods was the “asymmetric adjustment problem”:[1]

> “—In particular, Keynes (1942–3) held strongly the view that the major problem of all international monetary systems had been that they forced asymmetric balance-of-payments adjustment on deficit versus surplus countries: the former were forced to adjust, as they generally lacked adequate external financing or adequate reserves to manage crises, whereas surplus countries did not face similar pressures. Keynes’ obsession with this issue was, of course, related to the fact that this asymmetry generates a global contractionary bias during crises. [...] The asymmetric adjustment problem, therefore, continued to be a feature of the system designed at Bretton Woods as well as of the non-system that succeeded it.—”

In the absence of abuse, or in the presence of corrective measures, this built-in problem did not threaten the viability of the system.

The Bretton Woods period, from 1945 to 1971, saw unprecedented distributed economic growth, development of a strong Western middle-class, cultural, technological and scientific development, colonial liberation of Africa, and the creation of effective global negotiating bodies such as the United Nations, including several war-prevention protocols and conventions.

USA annuls Bretton Woods

The system worked too well. The USA experienced a growing deterioration of its preeminence as the main trade-surplus nation, and projected a difficulty in honouring the gold redeeming arrangement if confidence in the USA dollar were to falter. If the rules were maintained, the “asymmetric adjustment problem” could become a problem for the USA itself, which would be obliged to either reduce its large military expenditures or devastate its middle-class.

The first rule, written or unwritten, is that the boss can change the rules. “On August 15, 1971, without prior warning to the leaders of the other major capitalist powers, US president Nixon announced in a Sunday evening televised address to the nation that the US was [unilaterally] removing the gold backing from the dollar.”[2]

Nick Beams put it this way:[2]

> “—In 1971 an administration grouping under the leadership of Paul Volcker (later to become chairman of the US Federal Reserve Board) concluded that financing for US

> deficits has ‘permitted the United States to carry out heavy overseas military expenditure and to undertake other foreign commitments’ and that an important goal was to ‘free ... foreign policy from constraints imposed by weaknesses in the financial system.’ [...] Moreover, there was considerable support for the view within US ruling circles that if the system of controls on capital movements were scrapped, the US would be able to maintain its hegemonic position because of its weight within the world economy. Other nations would want to hold dollars because of the role it played in the international monetary system.—”

Unlocking the dollar from gold freed the USA to print as much money as it wanted, and to disregard any trade deficit (or “debt”) that it might accumulate, as long as the dollar kept its place as the *de facto* world currency. The mechanisms were brilliantly explained by Michael Hudson in his 1972 book “*Super Imperialism: The Economic Strategy of American Empire*”.

OPEC caught on quickly. The so-called oil crisis of the 1970s followed on the heels of the demise of Bretton Woods. Basically, the major oil-producing countries increased the US-dollar price of oil to maintain a constant gold-based value of oil when the dollar was decoupled from gold, thus protecting their true buying power despite oil-contracts in US dollars.[3]

Excluding gold itself, oil became the first global commodity to acquire a constant and significant value, as long as OPEC could control its price. No other essential legal commodity (labour, agriculture…) achieved this feat. Given oil’s central importance in the real economy, in industrial and domestic operations,[3] demand for the US dollar would be high and the US dollar would preserve its preeminence, as long as oil contracts were forced to be in US dollars.

This is why there is coercive military presence in the Middle East, and why Israel has become tied to the hip of the USA. It explains the destructive wars against Iraq and Libya, both oil-producing countries that sought freedom from the US dollar.

Another major global commodity that tends to be valued in “gold” prices is opioid drugs. The USA has an “existential” interest to ensure that opioid drugs are traded in USA dollars. This explains the USA occupation of Afghanistan (“In 2015 Afghanistan produced about 66% of the world's opium.”[4]), and the zeal with which the USA enforces drug patents and big-pharma monopoly. It explains the USA’s “war on drugs” in Latin America.

From a world-currency-based empire perspective, the USA war in Afghanistan is not an “error”, nor is it a “failure”: “After 16 years and $1tn spent, there is no end to the fighting – but western intervention has resulted in Afghanistan becoming the world’s first true narco-state.”[5]

Similarly, the “green revolution” of USA-patented GMO crops is an extortion racket for global agriculture, in which the seeds and tailored pesticides and herbicides are bought in US dollars.

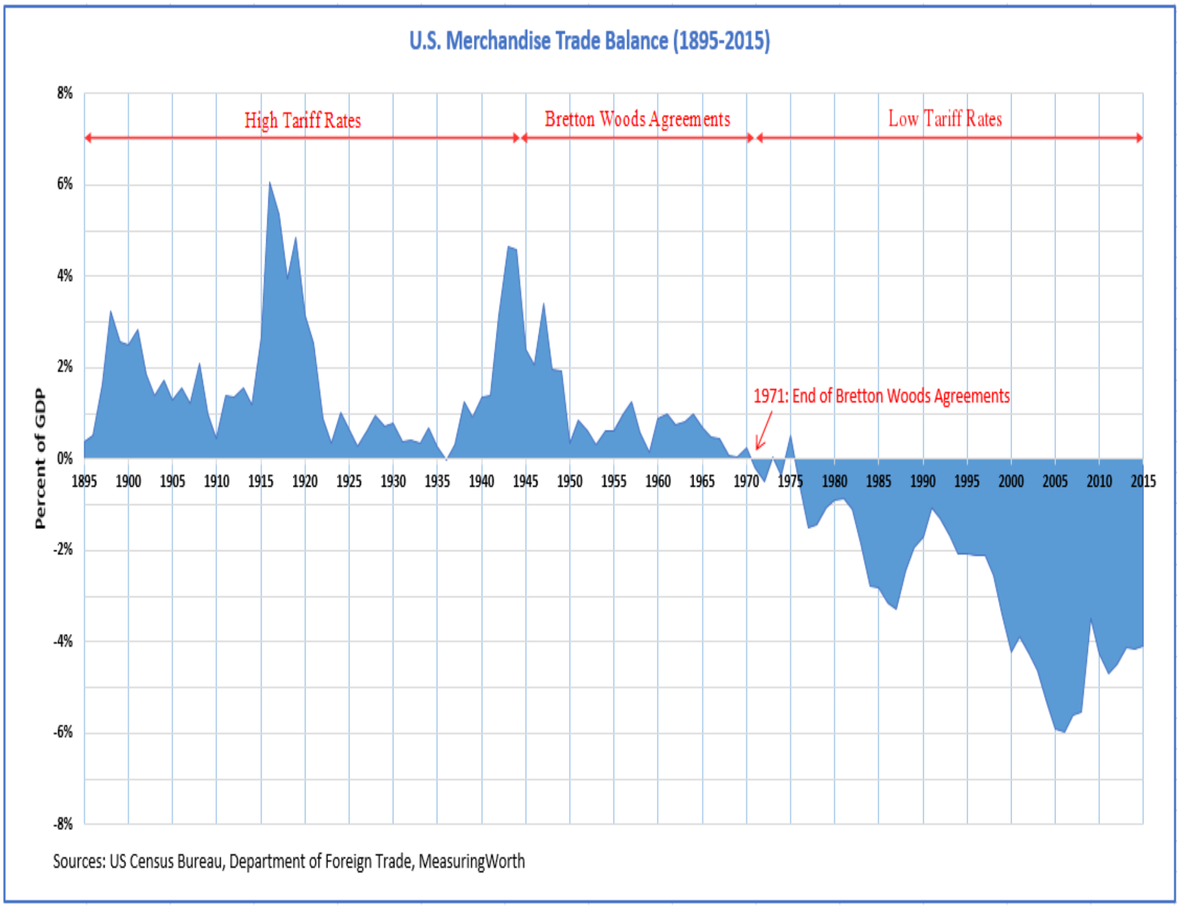
The ultimate over-priced item in the present non-system is USA military hardware sold, like any mafia sells “protection”, to all “allies” under the USA umbrella. To buy Russian military technology is a fatal or near-fatal transgression, as Saudi Arabia recently discovered with a contract for S-400 missiles, which almost caused a regime change.[6]

[3] Fossil fuels, today, comprise 87 % of all energy used in the world. Low tech (e.g., wood burning) and high tech (e.g., wind, nuclear) have proven to be either impractical in most settings or prohibitively expensive to manufacture, operate, and decommission (all activities requiring fossil fuel use).

Therefore, since the collapse of Bretton Woods, the USA has been forced to vigilantly project its military power to every continent, on aircraft carriers and through covert means, in order to impose the global currency that it prints. It is not uncommon for the USA to ship freight containers full of actual printed dollars to establish “democracy” in overthrown states such as Iraq.

The USA cannot be in every village and board room. Without the dollar, it is not the master of the world. This explains almost everything, as we will see further, below.

The most dramatic graph of the global economic significance of the Bretton Woods system and its demise is this one, showing the USA trade imbalance (as percentage of US-GDP) from 1895 to 2015:



The imbalance goes from positive and stable to negative, at 1971, and grows to a large permanent and negative value until this day. We also note a large increase in the negative slope at the 1991 dissolution of the Soviet Union, which is the main subject of this article.

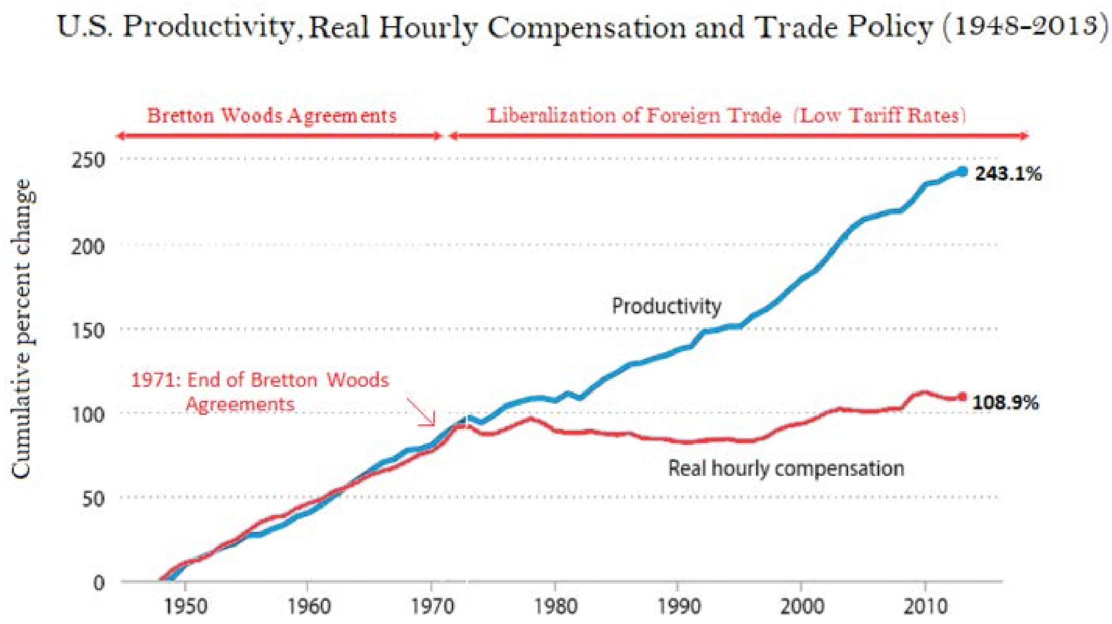
Societal impact associated with the collapse of Bretton Woods

USA power did not want its world dominance to be interfered with by economic constraints designed to provide global stability. It did not want to be limited in its military expenditures and it wanted to print the currency that would be imposed globally.

The USA exploited the 1970s “oil crisis” in institutionalized (media and academic expert) propaganda to justify putting a halt to middle-class development that had led to “The Crisis of Democracy”[7] and that had threatened its Vietnam and perpetual-war project.

It also criminalized and jailed the radical “ghetto” element of society in order to toxify the fertile ground that had produced Robert F. Williams, Malcolm X, the Black Panthers, and others. Urban ghettos would never again be allowed to independently be politicized.

These implementations are discernible in the following two graphs of macro-economic parameters. The first shows inflation-corrected hourly worker wages and USA productivity per hour worked, from 1948 to 2013:

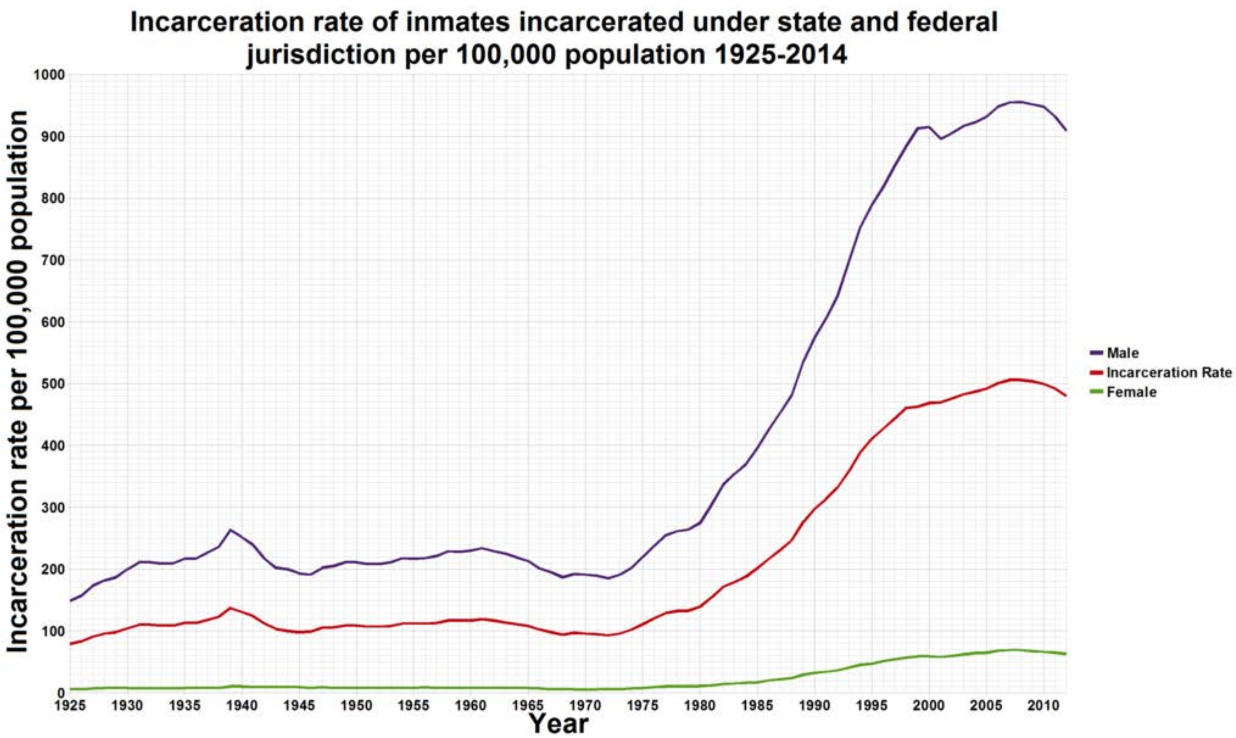


Note: Data are for average hourly compensation of production/nonsupervisory workers in the private sector and net productivity of the total economy. "Net productivity" is the growth of output of goods and services minus depreciation per hour worked.

Source: EPI analysis of data from the BEA and BLS (see technical appendix for more detailed information), Economic Policy Institute

The halting of worker wages is evident, and detaches from the increasing productivity per hour worked, following the USA’s cancellation of the Bretton Woods agreement (1971).

The second graph shows USA incarceration rates:



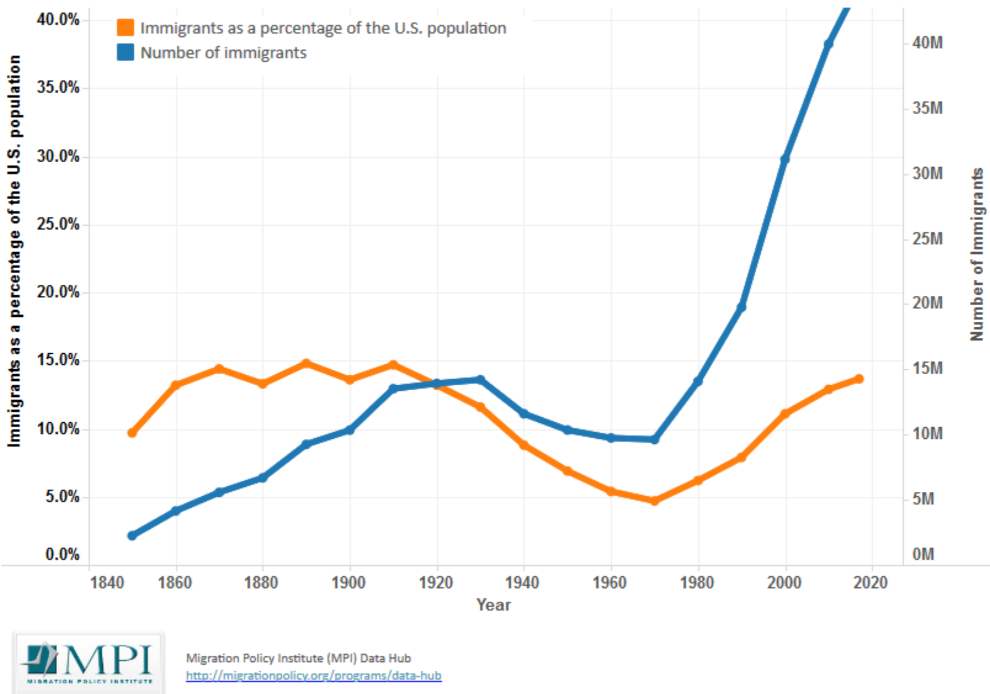
Starting at the sudden dismantling of the Bretton Woods system (1971), the incarceration rate for men increased five-fold, to a staggering near 1% of the male population. The devastation to communities plateaus at approximately the year 1998. The USA has the highest population incarceration rate in the world.[8]

These post-Bretton-Woods changes were accompanied by USA-managed finance globalization, which supports the US dollar as a global currency by increasing interlocked global trade and debt relations, and by enlisting the global elite:[1]

> "—The shift towards liberalizing capital flows started with the United States in 1974 but then spread to the rest of the developed world in the second half of the 1970s and through the 1980s, and was essentially completed by these countries in the early 1990s. […] This worldwide trend was reinforced by the multiplication and expansion of offshore financial centres. In any case, IMF rules continue to allow countries to regulate capital flows. The attempt by the managing director of the IMF, with US support (and pressure), to change the Articles of Agreement in 1997 to impose the obligation of capital account convertibility on Fund members was defeated. Major constraints on capital account regulation came with free trade agreements, notably those with the United States.—"

Post-Bretton-Woods globalization was not "free trade", nor was it "balanced development". It was anchored in USA control of the *de facto* global currency, and it was predatory. It eventually had palpable consequences in Western societies, beyond stagnation of real wages and out-of-sight incarcerations, such as the apparent homelessness in USA and Canadian cities, which accelerated as an emergent phenomenon in the 1980s,[9] associated with a predictable major Western recession—the 1982 crash, from Third World debt defaults on predatory loans from USA banks (see Michael Hudson's analyses). For an overview of the magnitude and duration of the global effects of the early-1980s recession, see the *Wikipedia* article "Early 1980s recession" (accessed on 28 February 2019).

The said post-Bretton-Woods predatory globalization gave rise to what could be termed "globalization migration". Although, to our knowledge, it has previously not been linked as such, a macro-economic or social-geography signature of the USA cancellation of the Bretton Woods system is the dramatic increase in legal immigration to the USA, starting in approximately 1971:[10]

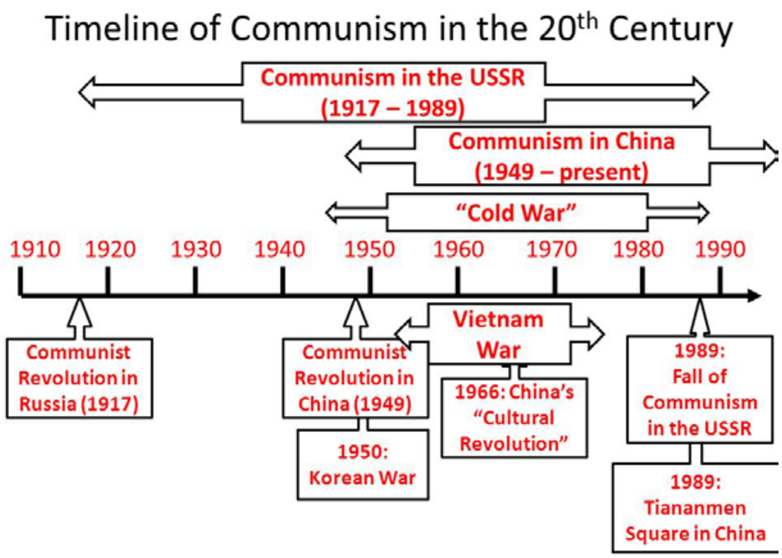


Thus, we see a post-WWII USA that first consolidated its strength by building up the "free world" to oppose the communist blocks, using elements of state-of-the-art macro-economic theory (Bretton Woods), then, in 1971, abandoned cooperation to preserve its top-predator status, suppressed dissent at home, and pursued covert and military enforcement and spread of world-currency-enabled exploitation by any means, except to the extent that it was limited by Cold War opposition.

This is the USA-centered context of the next global event that changed the world: the 1991 fall of the Soviet Union.

The 1991 fall of the Soviet Union

Following Russia's long, costly and transformative war in Afghanistan,[11] against CIA-armed jihadist resisters, the Soviet Union dissolved on December 26, 1991, by declared recognition of independence of the former Soviet republics. The fall of the Soviet Union was a major world event, comparable in importance to the world wars that preceded it.[11] This graph gives a world-communist perspective of the event:



Globalization response to the fall of the Soviet Union

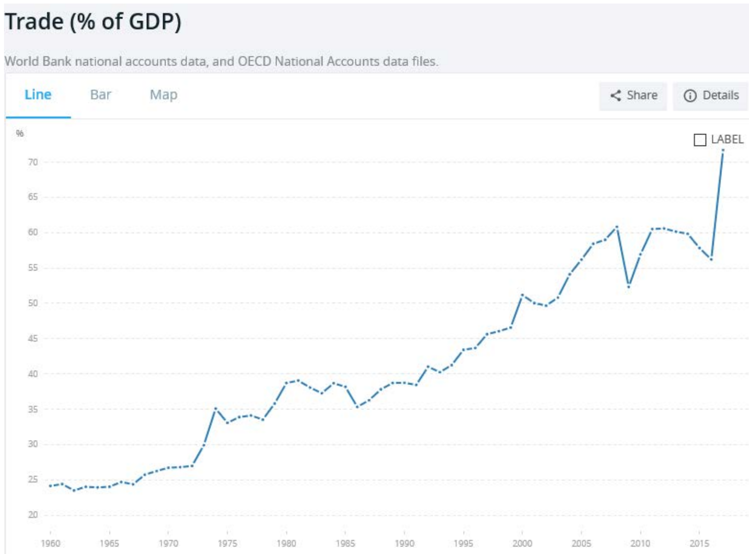
It is not generally recognized that the 1991 fall of the Soviet Union spurred accelerated "globalization" that restructured Western domestic societies to a degree comparable to the magnitude of changes occurring in a war period. But this fact is evident from many global parameters.

The circumstances that should have led to an increase in distributed global wealth from integrated military and economic cooperation with former Cold War foes, a la Bretton Woods, instead led to a USA rampage for unrestricted exploitation of and dominance over formerly protected regions.

The USA could not invent wars fast enough, to enforce its will and its currency: a renewed war on drugs, the Gulf War, wars to "prevent genocide", NATO expansion, the war on terror, wars to bring "democracy" and "human rights", war to "protect transportation routes", and so on.

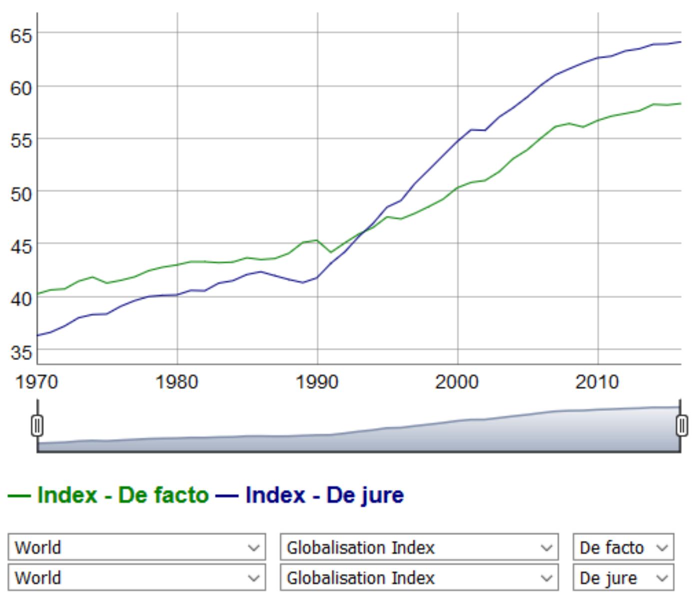
An aggressive financial "globalization" ensued. Investment revenues went through the ceiling and elite salaries became stratospheric, at the same time that targeted influential professional orders (teaching and civil service) were gutted of professional independence, and the support structures of the working class were decimated, including their jobs. In contrast, lawyers and doctors were elevated to the highest corporate-service levels.

Acceleration of *de facto* globalization is seen in a graph of world exports plus imports as percentage of GDP (gross domestic product):[12]



Here, we see a first rise following dissolution of the Bretton Woods system, a plateau up to the 1991 fall of the Soviet Union, followed by the current accelerated globalization (large positive slope).

The post-1991 acceleration of globalization is also recorded in the “KOF Globalization Index”:[13]

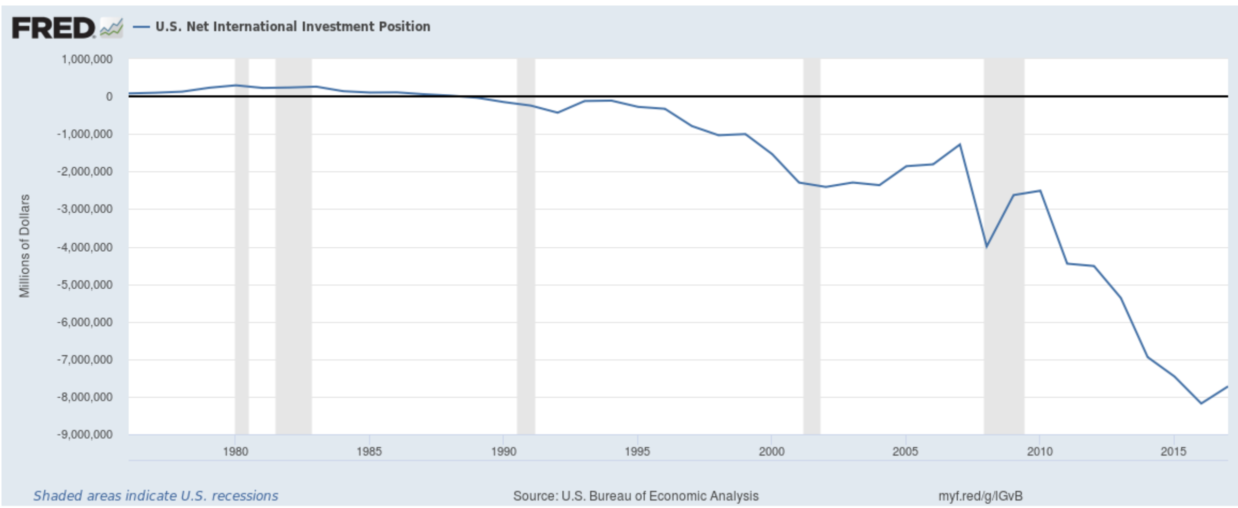


Here, “de facto globalization measures actual international flows and activities, de jure globalization measures policies and conditions that, in principle, enable, facilitate and foster flows and activities”.[13]

The Bank for International Settlements (BIS) describes the said post-1991 acceleration of globalization in the following terms:[14]

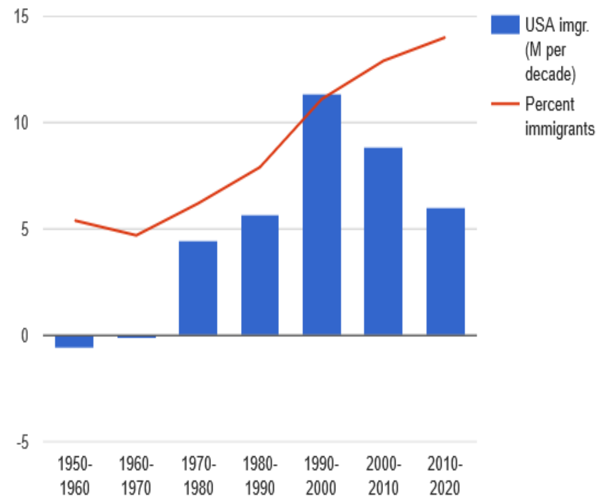
> "—External financial assets and liabilities have soared, from around 36% of GDP in 1960 to around 400% ($293 trillion) in 2015. The rapid expansion in financial openness from the mid-1990s has been concentrated in advanced economies. Relative to GDP, the external positions of advanced economies and emerging market economies (EMEs) were roughly equal up until the early 1990s. Since then, the cross-border financial assets and liabilities of advanced economies have surged, from roughly 135% to over 570% of GDP.—"

The post-Soviet-Union globalization, the post-1991 accelerated globalization, the new globalization, is a globalization era characterized by a large negative net international investment position (NIIP) of the USA, in which the USA became the largest debtor nation:[15]



See Wikipedia for a background definition of NIIP, and references therein.[16]

The post-Soviet-Union globalization (1991-), like the post-Bretton-Woods globalization (1971-) is highlighted by a large onset of USA immigration. In the following graph, we note large step-wise increases in the decadal changes in number of USA immigrants, occurring at the decadal markers 1970 and 1990:

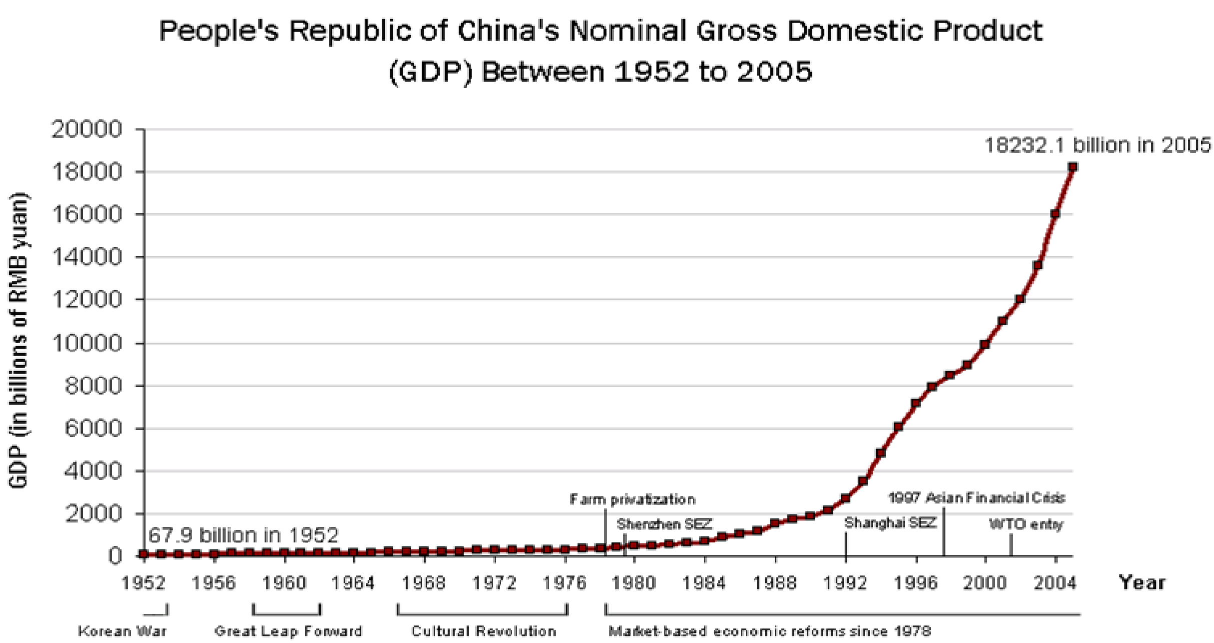


The decadal change in USA immigration population is virtually zero until the 1970-1980 decade in which it jumps to almost 5 million per decade, is virtually constant up to 1990, and then jumps to over 10 million per decade in 1990-2000.

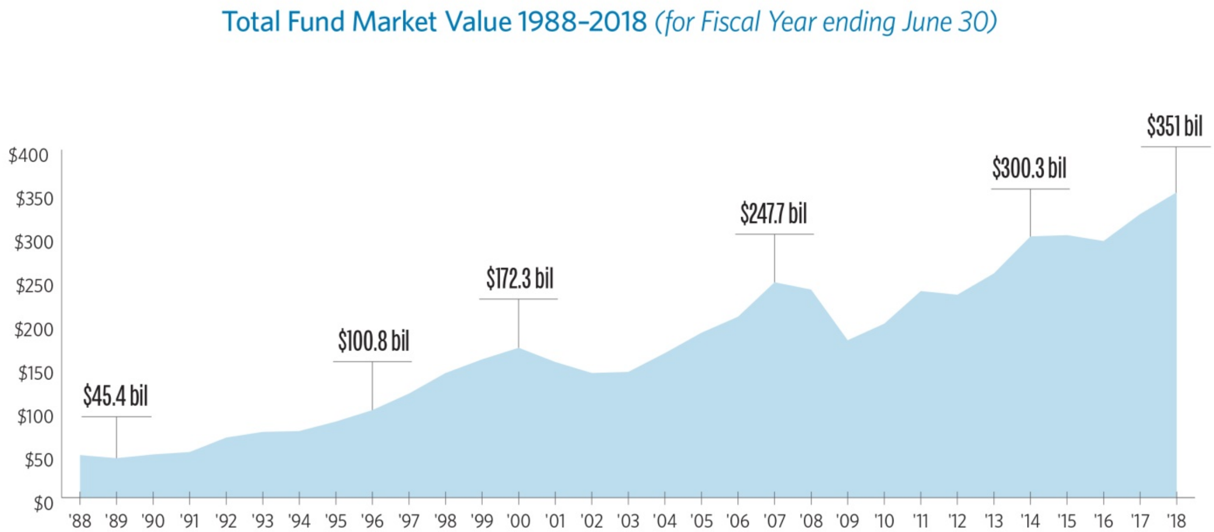
Here, the "percent immigrants" (red line) is the value at each end of decade. The yearly data counts all immigrants (persons not born in the USA), both legal and illegal. The values for 2010-2020 are linearly extrapolated from the data up to 2017. Source of the data: MPI.[10]

The post-1991 accelerated globalization is also seen in China's economic development. China was in-effect integrated into capitalist globalization at the 1991 turning point, as show in a graph of its GDP:[17]

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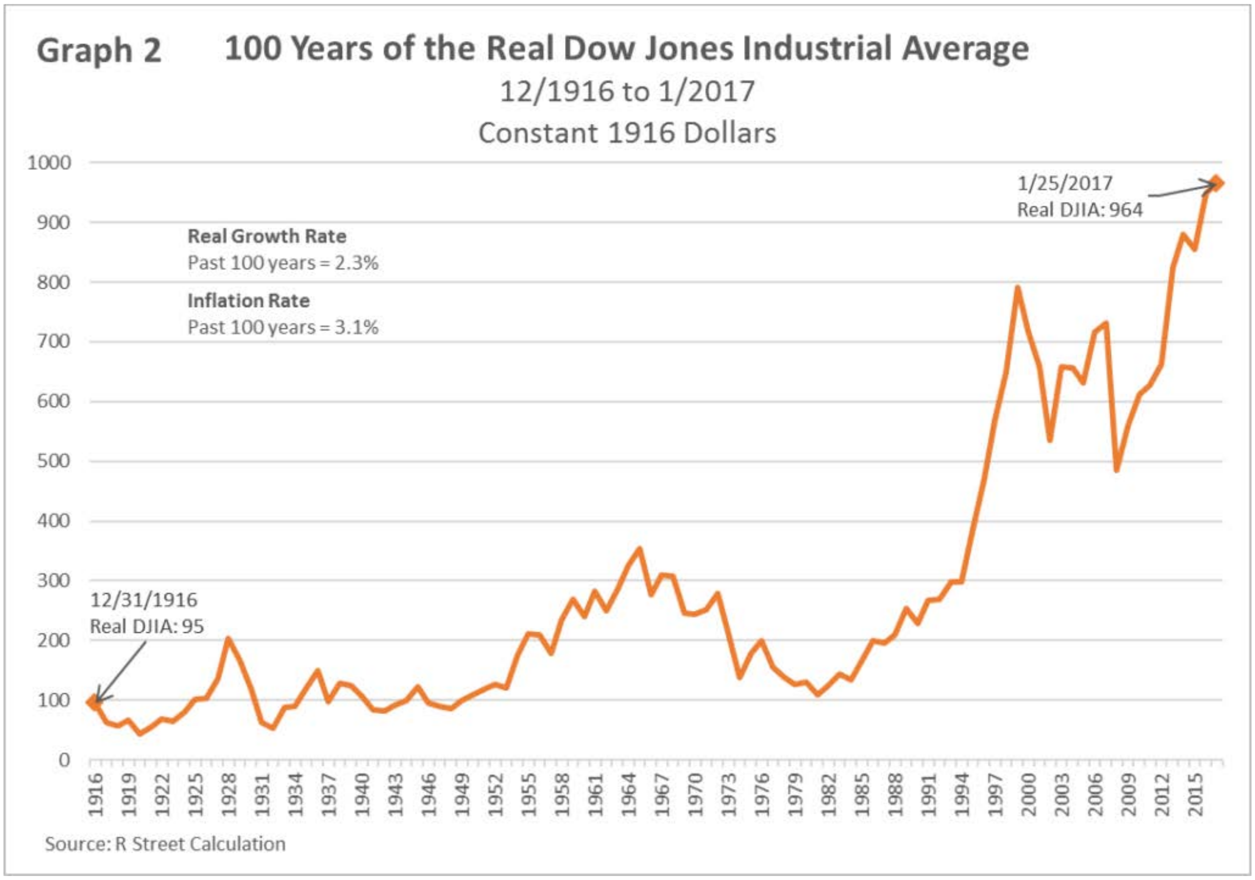


There are many records of the investor-benefits and elite-managerial bonanza that ensued. A conservative indicator of investment profits is seen in the California Public Employees' Retirement System (CalPERS) fund value evolution from 1988 to 2018:[18]



Here, a positive mean slope of 10 billion per year is sustained after 1991. “As of June 30, 2014, CalPERS managed the largest public pension fund in the United States, with $300.3 billion in assets” (Wikipedia).

The inflation-adjusted Dow Jones stock market index shows a more dramatic post-1991 rise of globalization, in one of its main effects:[19-a]



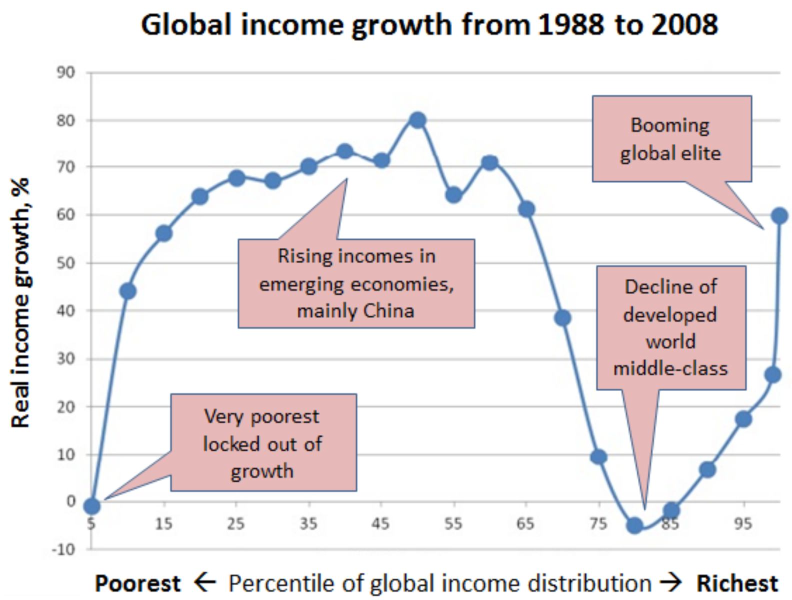
The initial lag, immediately following 1991, may be due to the lag-time for the USA military apparatus to position itself and to be perceived as "assertive" regarding globalization, in the post-Soviet-Union era. Regarding the 1995 Taiwan Strait crisis, BBC News put it this way: "The US' pivotal role was most clearly shown in 1996, when China conducted provocative missile tests to try and influence Taiwan's first direct presidential election. In response, US President Bill Clinton ordered the biggest display of US military power in Asia since the Vietnam War, sending ships to the Taiwan Strait, and a clear message to Beijing."[19-b]

However, the said initial lag, may be largely due to the time between initialing of the draft (October 1992) and establishment (January 1, 1994) of the massive North American Free Trade Agreement (NAFTA). It appears that the first task of the USA, following the fall of the Soviet Union was to strong-arm Mexico and Canada into the vast socio-economic integration that was NAFTA. The impact of NAFTA on Canadian society has been transformative.[20]

Effect of post-Soviet-Union globalization on social-class structure

The corresponding geographic class restructuring that has occurred since the post-1991 accelerated globalization has been described using the growth-by-income-percentile-distribution known as the "elephant graph".[21][22] The social geography of the changes in Western states has been studied in detail by Christophe Guilluy.[23] The ("elephant") graph of percent real income growth since 1988 (to 2008, or present, say) by percentile of global income (as a density distribution) shows a frozen poverty segment, rise of a middle-class in developing economies

(China), decline of the Western middle-class ("deplorables"), and disproportionate income growth of the managerial and elite classes ("bobos") as:[24]



The elephant graph can also be constructed for years from 1980 to 2016 and using a logarithmic scale for the higher income group percentiles, which looks like this:[25]

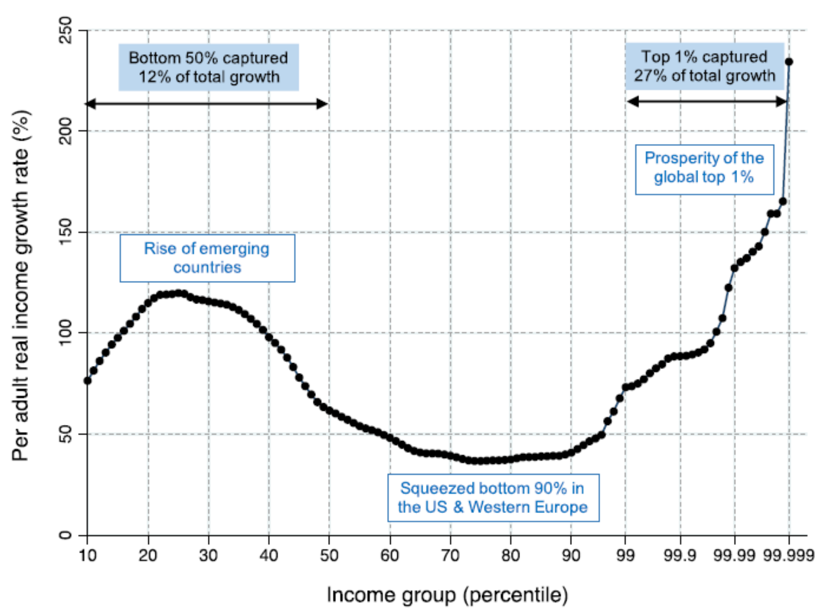
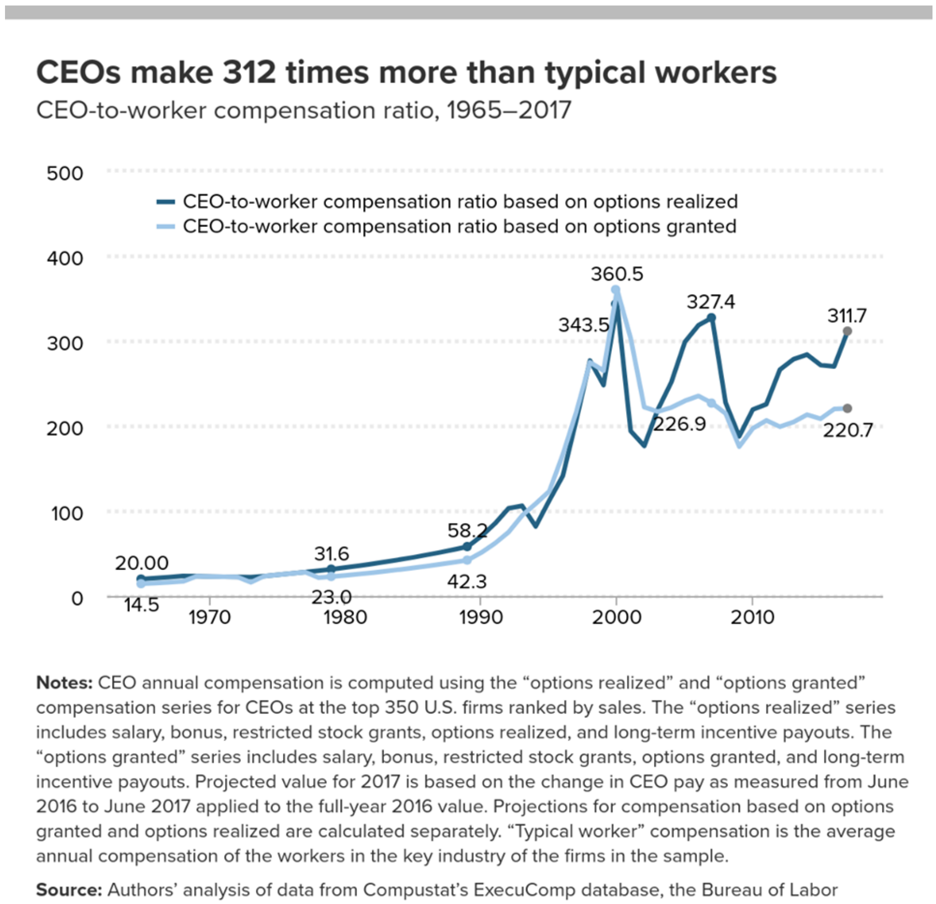


Figure 2. Total income growth by percentile across all world regions, 1980-2016

These "elephant graphs" reflect two other main features that characterize the new post-1991 globalization, beyond the coarse aspects of reported global trade and financial mobility. These are two sides of a campaign of class restructuring that created the present Western-class divide

between the “bobos” and the “deplorables”,[26] referred to as the “anywheres” and the “somewheres” by British journalist David Goodhart: rocketing elite salaries, and dismantling of the independent-professional and working-class support systems in Western nations.

The takeoff of “rocketing elite salaries” is well documented, such as in the ratio of CEO to worker incomes, which closely matches or correlates to global investment returns (see Dow Jones Index, above):[27]



Likewise, clear early-1990s rises in the incomes of top percentile income groups is detected in data up to the year 2000 for Canada and the USA, but was not linked by those authors to accelerating globalization.[28]

Emergent features in the new (post-Soviet-Union) globalization

Novel features in the new globalization included more aggressive predation of allied-country economies through new massive trade agreements, and through increased investment mega-mergers across national boundaries.

In North America, there was NAFTA, the North American Free Trade Agreement. It was wholesale dismantling of the trade and social-policy sovereignties of Canada and Mexico; a corporate and investor agreement in the hands of the dominant USA partner.

In Canada, the NAFTA trade agreement worked to dismantle social-program sovereignty, in at least two ways.

First, those domestic business "leaders" and interests that benefit from globalization relentlessly apply political pressure for the dismantling:[20]

> "—Since the beginning of the free-trade era, Canada's own business elite has argued that Canadian social programs would have to conform to the generally inferior U.S. levels in order to maintain competitiveness. As early as 1980, Laurent Thibault, who later became president of the Canadian Manufacturers Association, told a Senate committee: "It is a simple fact that, as we ask our industries to compete toe to toe with American industry ... we in Canada are obviously forced to create the same conditions in Canada that exist in the U.S., whether it is the unemployment insurance scheme, Workmens' (sic) Compensation, the cost of government, the level of taxation, whatever."
>
> Indeed in April of 1989, just four months after the implementation of the CUFTA, the Conservative government brought down what became known as its "free-trade budget". It included cuts to Unemployment Insurance, Old Age Security and federal transfers to provinces for health care and education. This pattern of spending cuts continued throughout the mandate of the Conservative government. It was accelerated after the Liberals were elected in 1993 and especially pronounced in the watershed budget of 1995 which included C$29 billion in spending cuts over three years.—" (at p. 53)

Second, the very principle of foreign investment works its magic:[20]

> "—The broader social impact of NAFTA is captured by Ken Traynor of the Canadian Environment Law Association who comments, "The old issue of who gets what even when overall 'economic efficiency' may have been enhanced is worth examining. Consider moving brassière manufacture from Cambridge, Ontario to Juárez on the Mexican border. $8 per hour wages paid to women in Cambridge to produce brassieres sold for $20...gets spent in the immediate vicinity of their homes, gets taxed and the firm generates local municipal taxes too. With NAFTA and a shift of production to the maquilas, only $2 of the $64 per day wages saved goes to the women in Mexico and almost none of the municipal and other taxes are paid in Mexico. The $62 per day per worker gets reallocated to Exxon for fuel to ship things around, to road transport companies, to brokers, and to the company itself and the spending circle of these guys is very different than that of the women displaced. And where the money circulates does matter," Traynor concludes, "especially to the women in this example."—" (at p. 55)

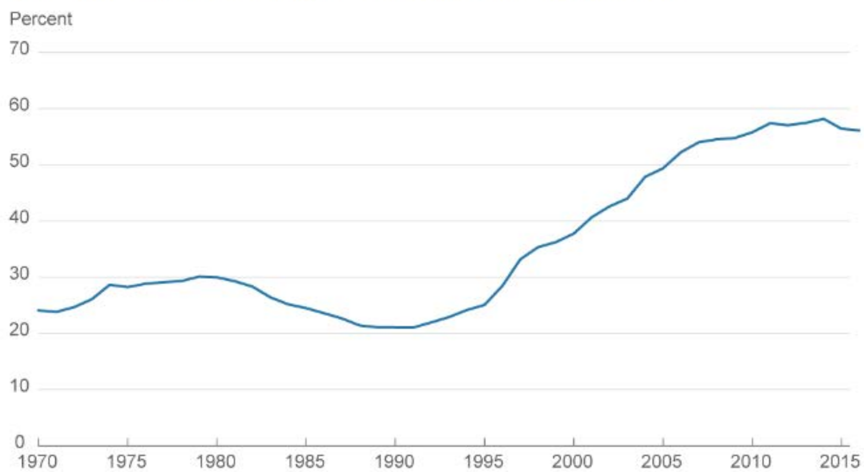
The said magic was well understood by all the great classic economic theorists of the industrial era but has been turned on its head in the false logic of post-Bretton-Woods finance globalization, and with a vengeance in post-Soviet-Union globalization.[29]

Indeed, the said vengeance is also seen in the other important emergent feature in the new globalization of the post-Soviet-Union era, which is the following.

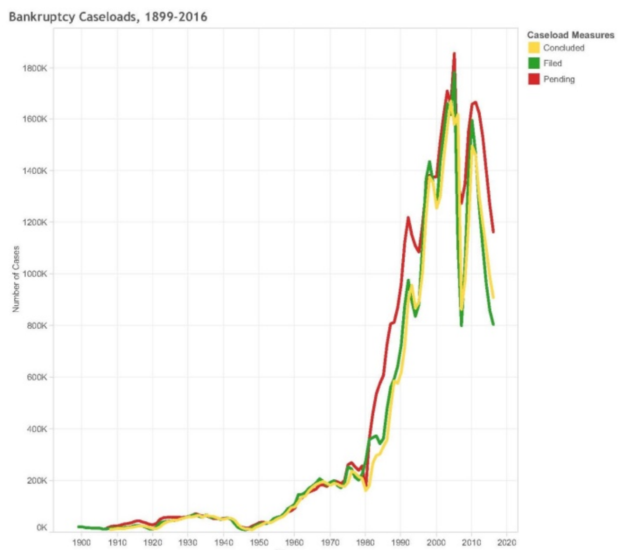
The USA predation is no longer solely based on the wealth-extraction conveyer belt of printing the imposed world currency. In addition, the large USA banks (Wall Street) have bought the Democratic Party outright and (in addition to their muscle in the USA system) have thereby acquired free reign to create money for themselves by scamming, and fixing and gaming the system, while being bailed out when the most daring mega-hustles fail, such as in 2008.[29]

The said emergent feature is well illustrated by this graph of percentage of total banking assets belonging to the ten largest (USA) banks:[30]

Figure 1. Percentage of Total Banking Assets Belonging to the Ten Largest Banks



This phenomenal concentration of capital ownership is accompanied by a large incidence of USA bankruptcies, under all judicial structures:[31]

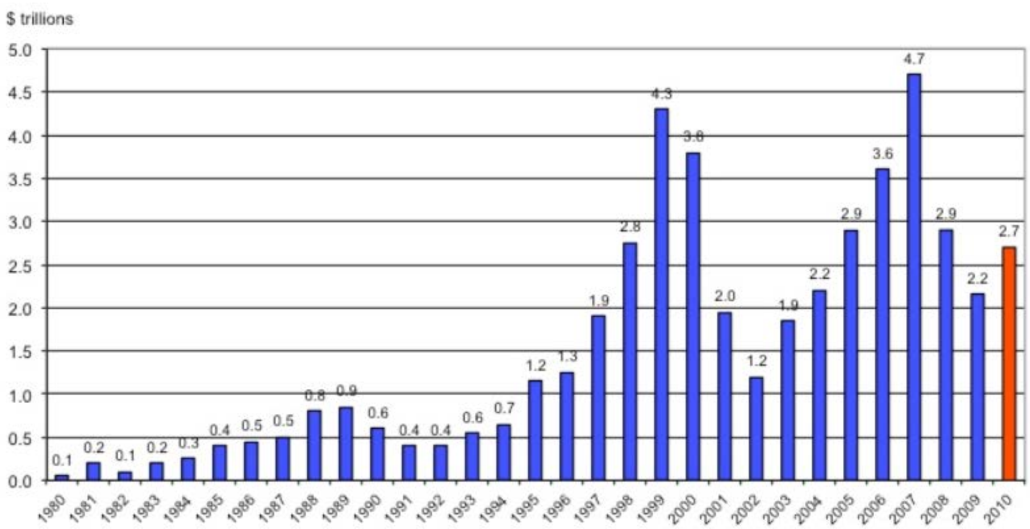


More concretely, the said emergent feature of the new globalization corresponds to an unprecedented wave of global mega-mergers, the so-called “fifth wave of mergers (1993-2000)”, in the economic history of mergers and acquisitions.[32] USA mergers lawyer Martin Lipton described the fifth wave this way:[33]

> “—**Fifth Period – 1993 to 2000.** This was the era of the mega-deal. It ended with the bursting of the Millennium Bubble and the great scandals, like Enron, which gave rise to the revolution in corporate governance that is continuing today. During the fifth wave companies of unprecedented size and global sweep were created on the assumption

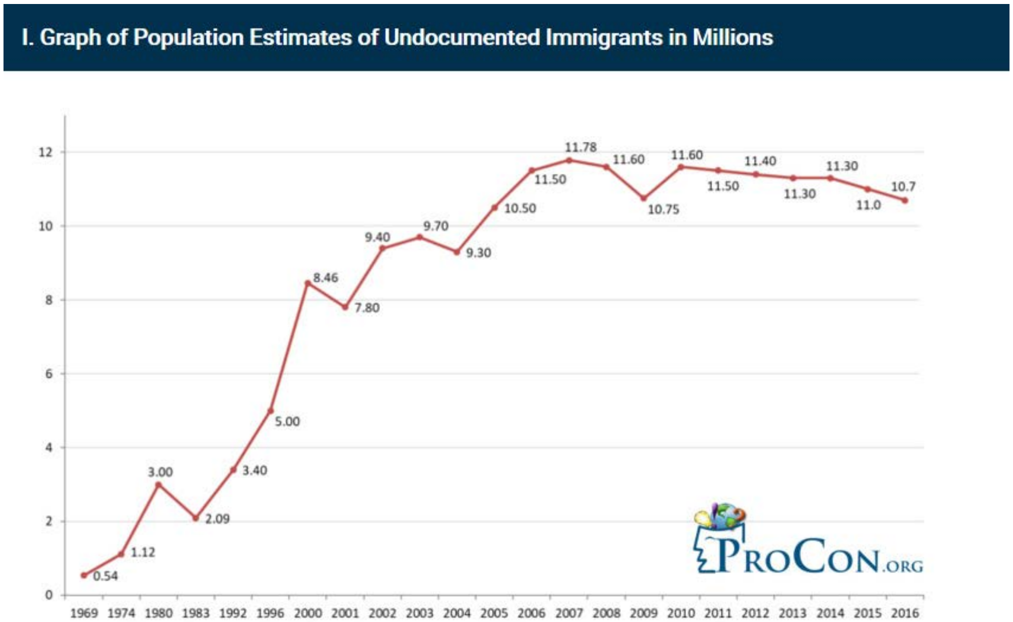
that size matters, a belief bolstered by market leaders' premium stock-market valuations. High stock prices simultaneously emboldened companies and pressured them to do deals to maintain heady trading multiples. A global view of competition, in which companies often find that they must be big to compete, and a relatively restrained antitrust environment led to once-unthinkable combinations, such as the mergers of Citibank and Travelers, Chrysler and Daimler Benz, Exxon and Mobil, Boeing and McDonnell Douglas, AOL and Time Warner, and Vodafone and Mannesmann. From a modest $342 billion of deals in 1992, the worldwide volume of mergers marched steadily upward to $3.3 trillion worldwide in 2000. Nine of the ten largest deals in history all took place in the three-year period 1998-2000, with the tenth in 2006.—"

This graph of volume of mergers per year (in trillions of dollars) is worth a thousand words:[34]



The onset of the fifth wave is ascribed to "deregulation and globalization",[32] however, we have not found any analysts or commentators who have linked the fifth wave of mergers (the new globalization) to the 1991 fall of the Soviet Union. It seems to us that the fall of the Soviet Union would have generated a zeal for USA-led deregulated mergers because the sudden absence of a global superpower-backed competing block would make capitalist satellite jurisdictions (Canada, Europe…) particularly vulnerable.

Indeed, the damage caused to Mexico by the new globalization (NAFTA and predatory investment) is strikingly reflected in the post-1991 surge of illegal USA immigration:[35]



Here, the number of undocumented immigrants shoots up in the mid-1990s, from values in the range 1-3 million to a plateau value of approximately 11 million, starting in the mid-2000s.

Post-Soviet-Union era down-scaling of the social safety net and human consequences of the increased globalization

The said "dismantling of the independent-professional and working-class support systems in Western nations" is difficult to illustrate by a single graph of some global economic parameter, because the changes were fragmented through several layers and regions of governance and different territorial jurisdictions (nation, state or province, municipality, public board). The changes were varied and occurred in institutions, regulations, statutes, governance structures, and management culture.

In Canada, the changes were experienced as though, in the vicinity of 1994 (NAFTA came into force on January 1, 1994), word was sent down from the governing elites, to every elected political leader, and to all influential board members of all the major public corporations, that everything needed to be overhauled because there was too much democracy for what was to come.

Regarding social transformation:[36]

> "—Up until 1995, provinces and territories received funding for their social programs through the Canada Assistance Plan (CAP), a policy introduced in 1966 that enshrined national standards for welfare policies and guaranteed matching federal funds for every dollar spent by provinces on social welfare programs, based on a core concept of 'need.'
>
> In 1995, as part of a debt reduction strategy, the federal government changed its formula to a block funding model, called the Canada Health and Social Transfer (CHST), which combined funding for health, education, and social welfare into one block transfer to each province and at the same time reduced national standards and the amount of funding that each province would receive.
>
> […] The overall number of people receiving social assistance fell from just over 3 million in 1993 to 1.75 million in 2003 (…) in spite of a growing population.—" (at pp. 37-38)

One end result, shown graphically, is in the number of social rental housing units completed per year:

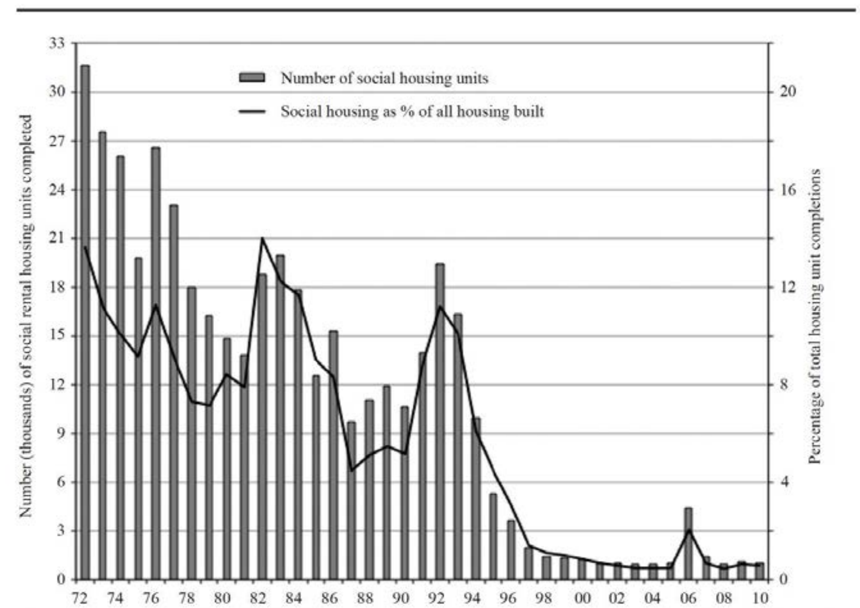
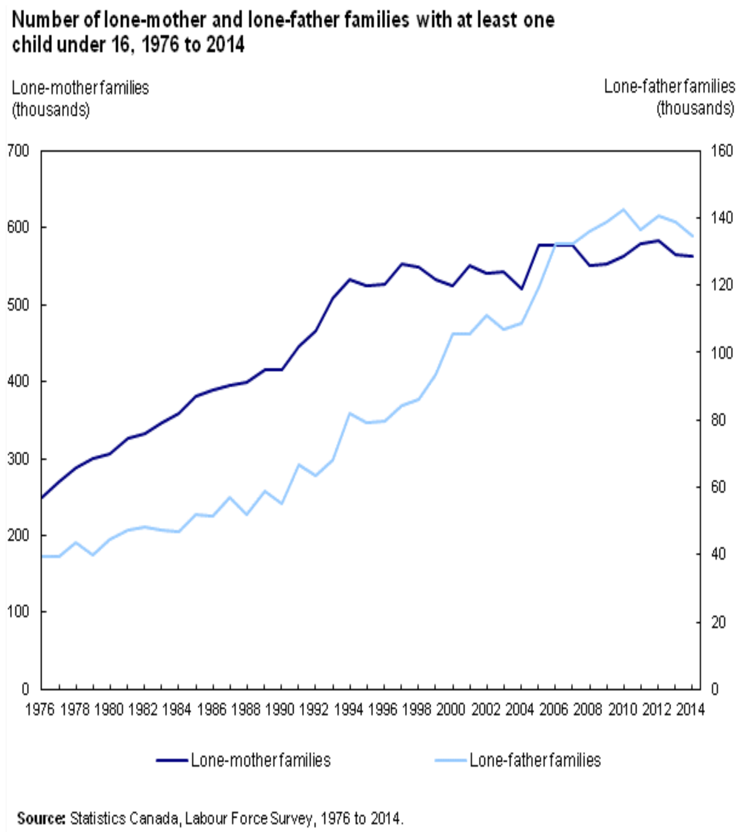


Figure 2. Social housing units built, by year, Canada 1972–2010 (source: calculated by the first author from CMHC *Canadian Housing Observer*, various years).

The number of lone-mothers with children rose from approximately 410,000 in 1990 to 530,000 in 1994. The post-NAFTA value of 550,000 lone-mothers is one-and-one-third the value at 1990, while the slope of increase in lone-fathers with children changed from 1,500 per year in 1976-1990 to 4,700 per year in 1990-2008:[37]



Therefore, the aggressive globalization wave that followed the 1991 dissolution of the Soviet Union may have put unprecedented strain on families with children. A strain on families is corroborated by the fact that, in Canada, the volumes of family-law and child-protection litigation in the courts increased dramatically (three-fold) from a relatively low quasi-plateau value in the 1980s, through a large climb in the 1990s, to a higher quasi-plateau value in the 2000s and 2010s. This is seen both in claims and applications received and in judgements rendered.[38]

In addition to the area of family law, court-judgement databases for Canada show 1990s threefold increases in numbers of judgements per year in several broad areas of law: criminal, bankruptcy, health, contract, and defamation.

A large increase (threefold, from 1993 to the early-2000s) also occurred in the USA, in terms of the legal needs of low-income households, as reported on the basis of several (nine) extensive surveys:[39]

> "—With one exception, all [nine] of the recent state studies found a level of [legal] need substantially higher than the level found in the 1994 ABA study. The ABA study found an annual average of 1.1 needs per low-income household, while the recent state studies range up to more than three legal needs per house-hold per year, as shown in Table 3. The ABA study thus represents the lowest figure available for estimating the number of legal needs experienced by low-income Americans.
>
> Footnote: The studies found that most problems were experienced in the areas of housing (such as evictions, foreclosure, and unsafe housing conditions), consumer (such as debt collection, bankruptcy, and consumer scams), and family (such as divorce,

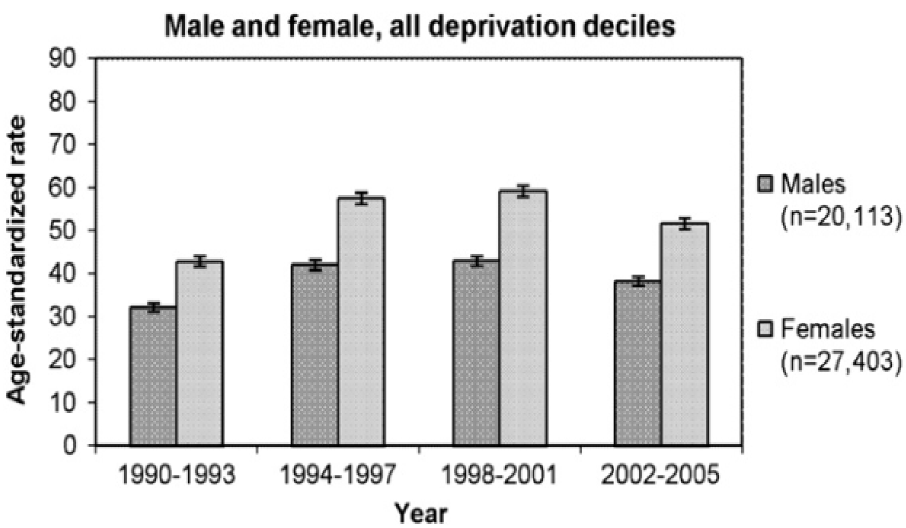
> domestic violence, child custody and support), as well as employment, government benefits, health care, and regional and community problems. Although the distribution of problem types varied somewhat from state to state, the same basic types of problems appeared in all nine states.
>
> Footnote: One recent state study found that the legal needs of low-income people in the state had actually increased over the preceding decade. The 2003 Massachusetts study documented a higher level of legal needs than had been found in a1993 state study using a similar methodology. The study concluded that these differences were not attributable to methodology, but rather indicated an increase in needs since 1993. —"

In our view, the 1990s increase in both need and litigation, occurring in several Western nations, is the fundamental cause of the widespread so-called "crisis in access to justice", although professional associations and legal commentators are silent on the increase, and have ascribed the "crisis" essentially to the high costs of lawyers.[40][41][42]

The full array of globalization-induced consequences to individuals and families in Western societies is expected to have included: job scarcity, job insecurity, lost prospects of job promotions, lower earnings, diminished State safety net, increased housing costs, increased travel costs, and so on.

These factors appear to have also translated into measurable impacts on individual health, including suicide, opioid overdose, and asthma, as follows.

Suicide attempt rates and mortality from suicide are established to be highly correlated with low economic status in Western societies; for example, in Quebec society (Canadian province).[43] The age-standardized rates of suicide-attempt hospitalizations (per 100,000 population) by gender, in Québec, show large significant increases in going from 1990-1993 to 1994-1997, of more than 30 %, for both genders:[43]



A 1990-1993 to 1994-1997 increase also occurs in the mortality rate from suicide, and large economic-status differences in suicide rates are seen for all age groups and both genders.[43]

The same type of trend occurs in the USA regarding suicide, such as with rates of hospital emergency department (ED) visits:[44]

> "—With regard to temporal trends, rates for attempted suicide and self-inflicted injury increased significantly over the 16-year period from 1993 to 2008 (P for trend <0.001). The average annual number of ED visits for suicide attempt and self-inflicted injury more than doubled from 244,000 between 1993–1996 to 538,000 between 2005–2008 (Figure 2), a ratio of 2.21 (95%CI; 2.02–2.40). […]
>
> This increase in self-harm visit rates per 1,000 US population was seen in all major demographic groups. Comparing 1993–1996 to 2005–2008, rates nearly doubled for both males (0.84 to 1.62) and females (1.04 to 1.96). Similar increases were noted for patients aged 15–19 (2.57 to 4.53), 30–49 (1.29 to 2.49), and those over 50 (0.11 to 0.90). Likewise, increases were observed for whites (0.94 to 1.82) and blacks (1.14 to 2.10).—"

This confirmed a similar earlier study, which was for the period 1992-2001.[45]

Opioid overdose population statistics show the same picture:[46]

> "—In a nationally representative database of U.S. ED [emergency department] visits, we found that the ED visit rate for opioid overdose quadrupled from 1993 to 2010. […]
>
> Between 1993 and 2010, the national ED visit rate for opioid overdose increased from 7 to 27 per 100,000 population (307% increase; Ptrend = 0.03) and from 19 to 63 per 100,000 ED visits (235% increase; Ptrend < 0.001—"

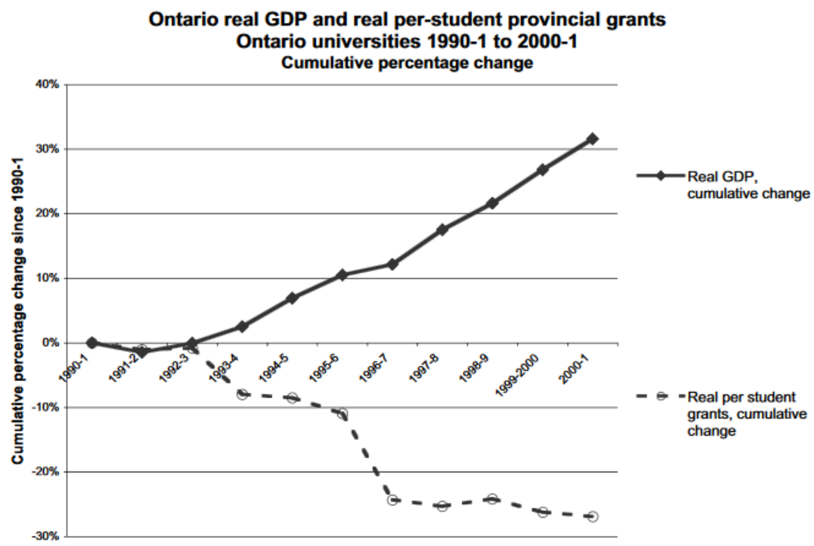
Asthma epidemiology is likewise informative. "Asthma is one of the most common chronic conditions affecting both children and adults."[47] We are said to be in the midst of a global asthma epidemic. In addition to genetic and environmental and other causal factors, there is a link to socio-economic stress: "Children whose caregivers report high levels of stress and who have difficulties parenting are at greatest risk for asthma."[47]

An authoritative review has:[48]

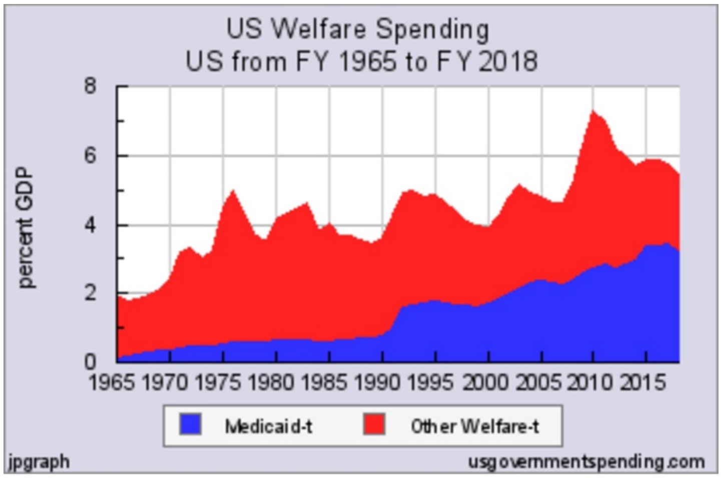
> "—The prevalence and incidence of asthma are very high in the Western world. […] According to the Centers for Disease Control and Prevention, the prevalence of asthma among U.S. children increased from 3.6% in 1980 to 5.8% in 2003. Asthma is the third leading cause of hospitalization among persons under 18 years of age in the United States, exceeded only by pneumonia and injuries.—"

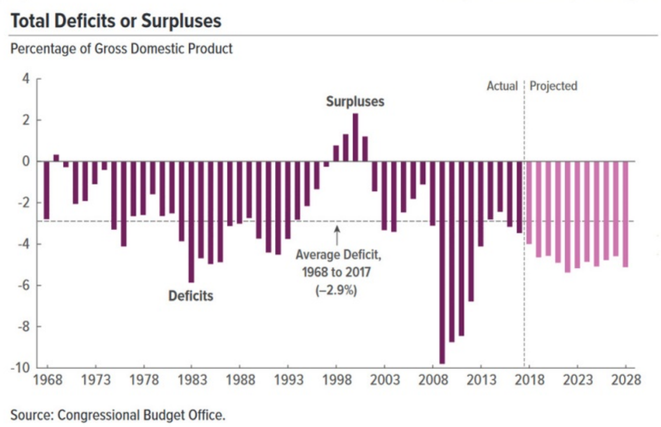
In the early-1990s, the number of ambulatory visits for asthma per 1,000 children 0–17 years of age in the USA rose from a 1980s plateau value of approximately 30 to values of approximately 60, extending into the 2000s (Fig. 3 in [49], and see Fig. 3 in [50]). At the same time, in the vicinity of 1993, the number of attributed asthma deaths in the USA, per 100,000 population, had a 17 % step-wise increase from approximately 48 to approximately 56 (Fig. 4, in [51]).

Coming back to NAFTA in Canada, in another example, the grants to university students were cut dramatically in Canada's largest province of Ontario, at the same time that the province's GDP grew equally dramatically:[52]

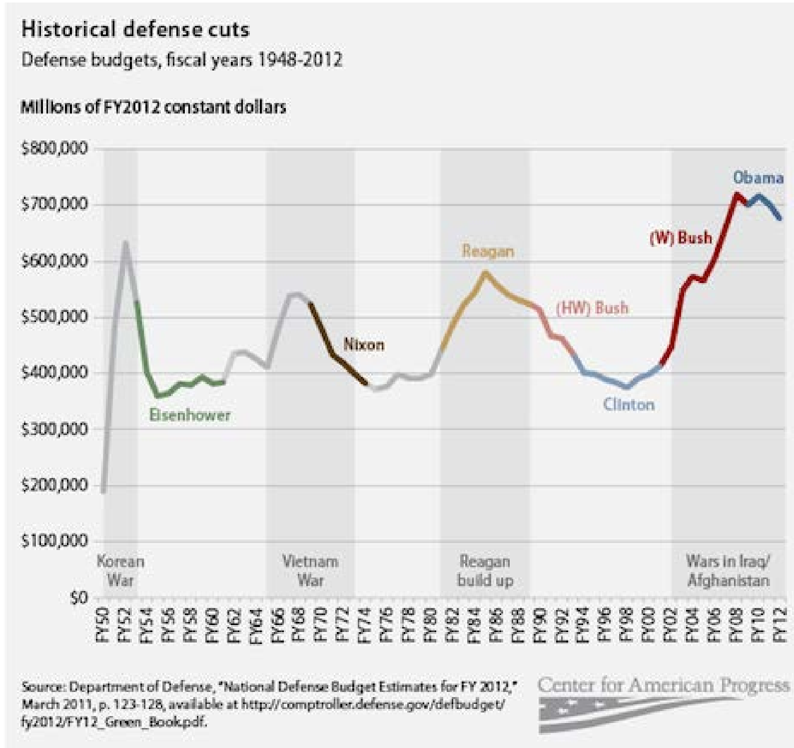


Such social cuts that followed the dissolution of the Soviet Union were not simply a consequence of adjusting to a more competitive trading partner. They seemed to be ideological, without a valid economic justification in terms of domestic strength, stability, and equity. For example, in the USA, President Bill Clinton (January 1993 to 2001) oversaw a massive cut in welfare, which produced the only federal budget surplus of the USA since the end of Bretton Woods, during a period of fairly constant defence spending:





For comparison, USA defence spending in the periods of globalization looks like this:



Removing professional independence by restructuring institutions

It was not just welfare that was cut in Canada. Large cuts and imposed structural changes were implemented in public education, in the research-university system, and in the federal civil service. The restructuring significantly reduced profession independence and academic freedom in these sectors.

Premier Mike Harris in Ontario (1995-2002) applied his so-called "common sense revolution", whereby he reduced primary school and high-school to holding and obedience-training farms by attacking the preparation and professional-development allocations of teachers. Teachers were reduced to overworked baby-sitters "delivering" a more centralized and directed curriculum. Grade-13 was eliminated altogether, without effective recovery at the university level. Student tolerance to Power Point presentations and sterile content increased. Undergraduate education in Ontario has never recovered.

The universities themselves were restructured, using draconian and arbitrary cuts. The Ontario Confederation of University Faculty Associations (OCUFA) reported:[53]

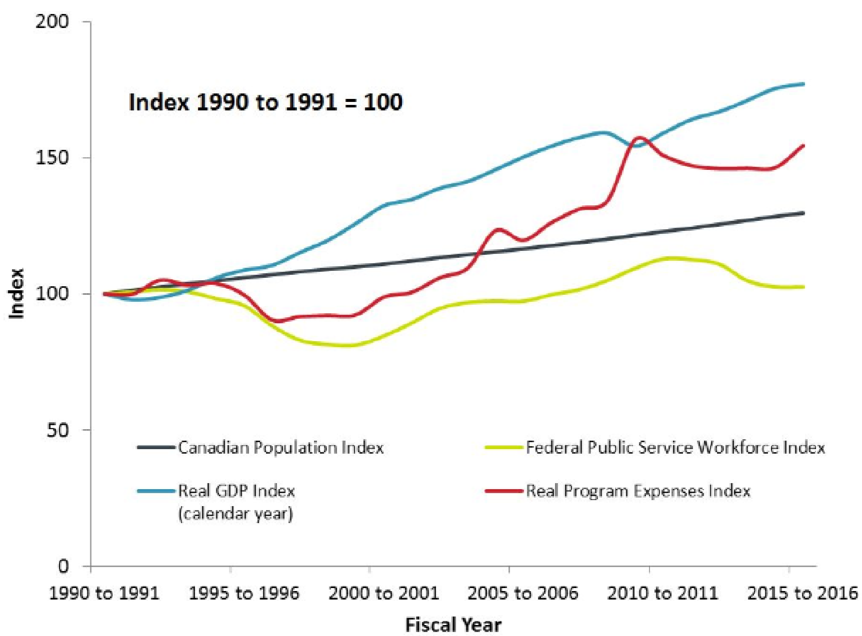
> "—Ontario Finance Minister Ernie Eves announced a 1 per cent cut to university funding - the full amount promised in the Tory Common Sense Revolution for the life of the government - as part of his November 29, 199 economic statement.—"

In response, for example, the University of Ottawa named an out-of-faculty hatchet-man dean of science who threatened to eliminate entire departments in order to get cooperation for increased centralization and resource reduction. Independent departments historically managed by committees of professors and students became centrally managed, and lost control over

their operating budgets. New staff was *de facto* named by the university administration, following centralizing strategic “mission statements”, rather than selected by the department. And so on. The net effect was that professors were further constrained to “their” research areas, and curricula were more centralized and regulated. Professors lost management of the academic units. Both professional independence and academic freedom were significantly reduced in the new organizational structure.

At the same time, the university research federal funding model was overhauled away from principle-investigator-directed independent research and towards industry-partnered research, and formalized mega-research alliances. University offices of industrial and contract cooperation mushroomed, at the same time that curricula became bland textbook-supported content delivered in a standardized way. Fewer and fewer tenured faculty were hired; and, in practice, a shrinking fraction of the research professors could tailor their research to supplement teaching, digress from the curricular track, or find the time to prepare classes from multiple and current sources. In any case, the students, coming out of the new high-schools, wanted only to be rewarded for obedience.

On the larger scale of the entire country, the new globalization that followed the 1991 fall of the Soviet Union, and the 1994 establishment of NAFTA, is seen in large cuts, from 1993 to 2000, to both the federal program expenses and the Canadian federal public workforce:[54]



The cuts in the federal government workforce were not uniform. Rather they targeted sector managers who had institutional memory and influence on policy, the so-called Assistant Deputy Ministers (ADMs) (“the most senior public servants appointed under the *Public Service Employment Act*”).[55] The ADMs are the largest contingency of high-level managers who attend Executive Committee meetings of the government ministries. They are the most influential government employees below the Deputy Minister and Associate Deputy Minister.

Most analysts present the social-services cuts as necessary to reduce the government deficit,[56][57] but otherwise outside of the historic global context. However, the actual cuts were preceded by a structural overhaul of the public service, intended to align and redesign the

government machinery in the new era of NAFTA. Prime Minister Mulroney had given Canada NAFTA, and then he resigned and gave the job of cutting and transforming government in order to implement NAFTA to Kim Campbell. She dramatically and irreversibly initiated the transformation in the four months of her only term in office as Prime Minister (July-October 1993), thereby preparing the government apparatus for the actual cuts to social services that would be made by the Liberal government.

Analyst Evert Lindquist describes the Campbell-implemented transformation in the following terms:[58]

> “— […] a bolt out of the blue which reduced the number of ministers and departments from 32 to 23, and affecting tens and tens of thousands of federal public servants. It was a comprehensive, fundamental re-design of the structure of the Canadian government, affecting not only the size and operation of Cabinet, but also the size and portfolios of a host of departments and portfolios. Planned in secret out with the Machinery of Government group in the Privy Council Office, the restructuring initiative had the general endorsement of Prime Ministers Brian Mulroney and Kim Campbell […]
>
> But the June 1993 restructuring, which was followed by the significant targets set out early on in the 1994-95 Program Review process, led to significant upheavals for public servants at all levels – gone was the long-held assumption that the Canadian Public Service could provide jobs and careers for life, since even high-performing individuals found themselves dismissed from their positions, in temporary assignments, or waiting for whatever positions would open up in the new departments (Lindquist and Paquet 2000). This break from long-held understandings was reinforced over the next decade with public servants increasingly receiving less protection from the media and more blame from governments and their ministers (Savoie 2003).
>
> It is also important to understand that the two-step sequence was not an accident: the designers of the June 1993 restructuring knew that when a new government was elected in late 1993, it would have to deal decisively with Canada’s growing deficit and debt situation and that difficult policy and program decisions would need to be made to deal with short-term needs, such as building confidence in financial markets, and longer-term rethinking of policies and programs in almost every sector. Restructuring the government at the level of cabinet and ministerial portfolios was seen as the means for repositioning the cabinet and its public service to make and implement these impending decisions.—”

In our words, constrained by NAFTA, you can cut social services rather than increase corporate and resource-extraction taxes, and reduce democratic participation in or control of society, but you need compliance to get it through: independent-minded civil servants who have an institutional culture of serving the public, and who have some sway, need to be brought in line. It’s the “difficult” ADMs that were let go.

To be clear, the historically unprecedented 1993-2000 cuts in “federal program expenses” (social-support programs) were particularly harsh and contrary to Canadian social-program design sovereignty, in that equivalent services or programs could not easily be recovered later, due to the Chapter-11 “investor rights” clauses of NAFTA. That is, foreign corporations would have to be compensated for future lost profits argued to arise from any recovery of sovereignty, with additional great legal expense in the litigation itself (and potential for public exposure).[20][59]

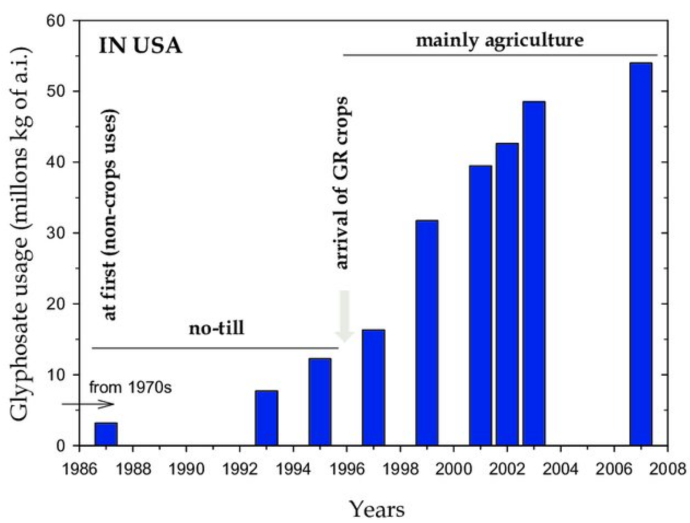
Post-Soviet-Union globalization deregulation of the agricultural and public-health industries

Following intense industry lobbying of USA scientists and politicians in the 1980s, government institutional culture of industry-compliance, and deregulation, accelerated in the early 1990s, setting the scene for global-market invasion of genetically modified crops (GM crops).[60] This USA phenomenon was followed in Western nations, after the regulatory principle of "substantial equivalence" was introduced by the UN Organization for Economic Cooperation and Development (OECD) in 1993, and endorsed by the UN Food and Agriculture Organization (FAO) and World Health Organization (WHO) in 1996.[61] Much resistance was offered by principled government scientists, but these efforts were overwhelmed.[62] Commercial sale of GM food started in 1994.

With such effective industry pressure to deregulate, and given the accelerated post-Soviet-Union (post-1991) globalization, new markets were created that transformed the agriculture, food, and public-health realms. This occurred with GM crops, pesticide use, and vaccines. All three had surges clearly starting in the early 1990s.

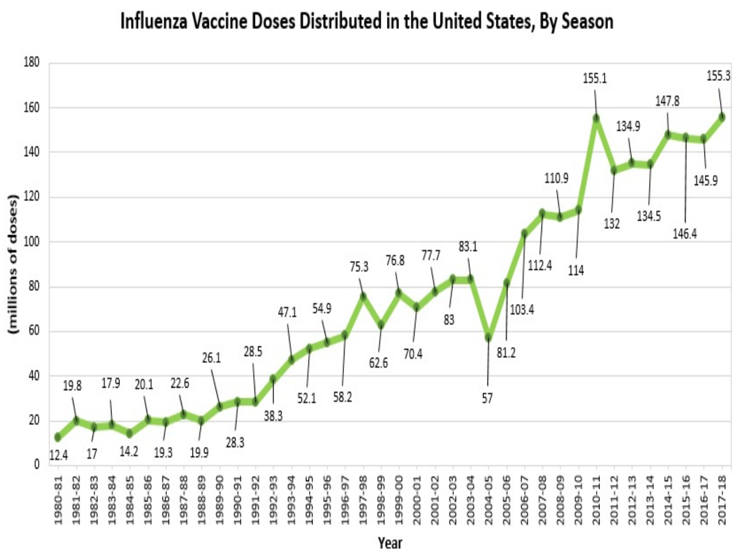
Regarding vaccines, the USA *National Childhood Vaccine Injury Act* (NCVIA) was enacted in 1986 and its National Vaccine Injury Compensation Program (NVICP) became operational in 1988. The program shielded the vaccine industry from liability litigation. It is a national limited-liability public litigation-insurance program, with its own claims tribunal. Since 1988, it has received over 20 thousand claims, and paid out approximately 4 billion US dollars to over 6 thousand determined victims of vaccine injury. The corresponding average risk of determined injury is approximately 1 in 1 million vaccine doses.[63]

Roundup is a glyphosate-based herbicide introduced by Monsanto in 1976. Nonetheless, the increased use of glyphosate did not start until 1993, but prior to the 1996 introduction of GM crops. Glyphosate is used in massive quantities, especially now in combination with GM crops, which are modified to be glyphosate-resistant, and which are imposed globally by the USA development-fund loan managers. The heavy use of the herbicide has caused glyphosate-resistant "super weeds" to emerge, so that even more glyphosate is used than would otherwise be needed. Glyphosate use shows a typical signature of post-Soviet-Union globalization, by its chronology and decadal monotonic increase (here "a.i." stands for "active ingredient"):[64]



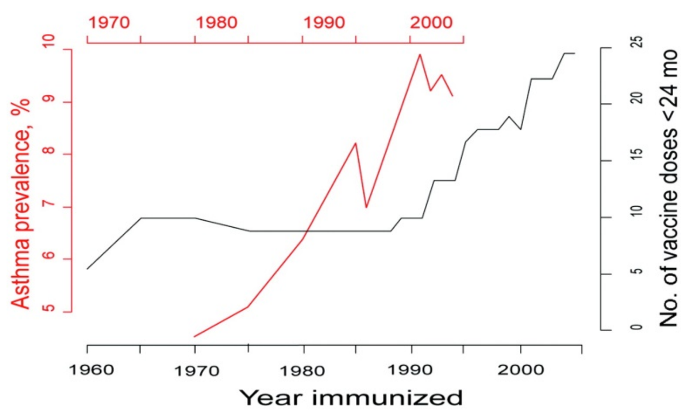
See also [65].

The number of seasonal influenza vaccine doses distributed in the USA is another example, showing the typical signature of post-Soviet-Union globalization:[66]



The influenza vaccine is overwhelmingly the most distributed vaccine in the USA, and has an average (1988-2018) NVICP-filed-injury rate of 3.3 per million doses.[63]

The number of vaccine doses delivered to less-than-24-month-old infants, per infant in the USA, also shows the typical signature of post-Soviet-Union globalization. It rose threefold, in the years from pre-1990s to 2005 (black curve):[67]



Spectrum of post-Soviet-Union globalization upsurges of chronic diseases

The USA databases of the Centers for Disease Control (CDC), and of the Surveillance, Epidemiology, and End Results (SEER) Program of the National Cancer Institute, show that there is a spectrum of chronic diseases, and some cancers, which have death rates and

incidence rates with typical temporal-trend signatures of post-Soviet-Union globalization. This spectrum of diseases has significant and sustained occurrence-rate increases, clearly starting in the early 1990s. The spectrum includes (and see the case of asthma, described above in relation to family stress):

- death from intestinal infections
- incidence of thyroid cancer
- death from Parkinson's disease
- prevalence of diabetes
- autism in children of different age groups
- phobia, anxiety disorder, panic disorder

One highly-cited research group has ascribed the cause to glyphosate toxicity, on the basis of documented correlation and plausible bio-molecular mechanisms.[68][69][70] Here are two examples of their graphical comparisons, for intestinal infection, and thyroid cancer:

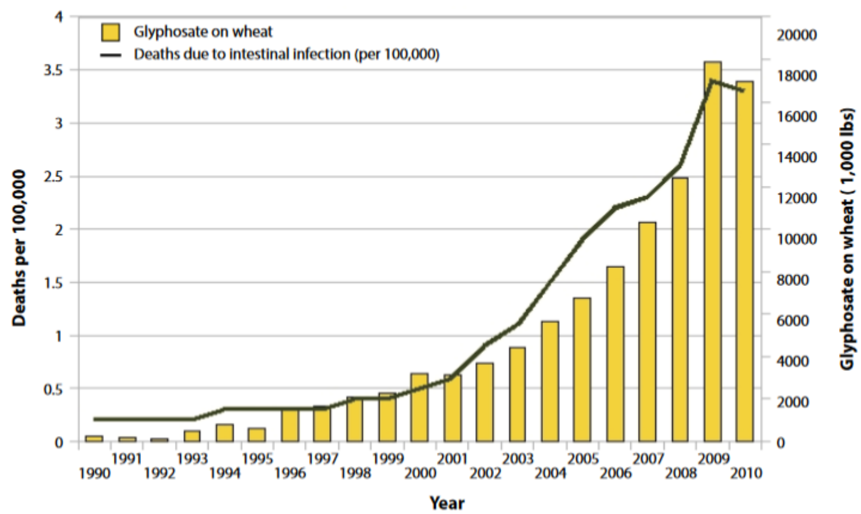


Figure 2. Deaths due to intestinal infections ICD A04, A09; 008, 009 with glyphosate applications to wheat (R=0.9834, $p \leq 3.975e\text{-}09$). Sources: USDA:NASS; CDC. (Figure courtesy of Nancy Swanson).

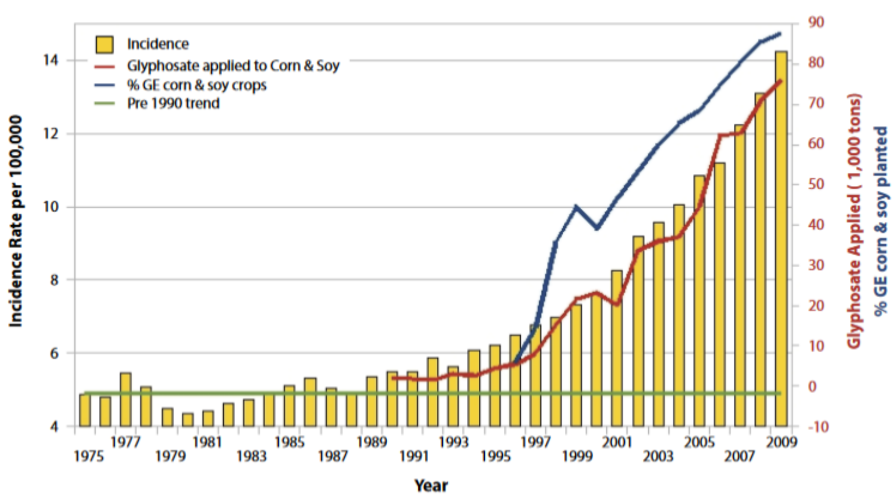


Figure 3. Thyroid cancer incidence rate plotted against glyphosate applied to U.S. corn & soy crops (R=0.988, $p \leq 7.612e\text{-}09$) along with %GE corn & soy crops (R=0.9377, $p \leq 2.152e\text{-}05$). Sources: USDA:NASS; SEER. (Figure courtesy of Nancy Swanson).

Summary of the new globalization in the post-Soviet-Union world

The fall of the Soviet Union was immediately followed by an accelerated globalization in which the USA dominated its “allies” for investment supremacy, became more predatory, and allowed its financial class more latitude to defraud than ever before since WWII.

European nations anticipated the threat and signed the Maastricht Treaty on February 7, 1992, which formed the European Union and led to a unified currency for protection of Europe itself.

Major features of the new globalization were unprecedented mergers in the finance, agri-food, pharmaceutical and information-technology sectors, aggressive investor-centered “free-trade” pacts, rise of the global elite and its entourage, loss of socio-economic security for the Western middle-class, concurrent negative public-health impact (suicide, emergency-department visits, asthma), increased leniency in food and drug regulation, concurrent upsurge of disease and chronic ailments (death from intestinal infections; incidence of thyroid cancer; death from Parkinson’s disease; prevalence of diabetes; autism in children of different age groups; phobia, anxiety disorder, panic disorder), integration of China into the capitalist finance sphere, accelerated and unprecedented USA negative trade balance, alignment of the top-layer service professionals and intellectuals into the paradigm and application of the new globalism, and an increase in USA global military presence and unilateral war campaigns (first NATO, then post-9/11).

The post-9/11 war campaigns protected the US dollar from abandonment, showcased USA military strength and aggressiveness, destroyed nations seeking sovereignty from USA dominance, secured the opium trade, increased control over oil, frustrated Eurasian integration, created CIA-managed terrorist proxies from the devastation of war, and created a strong demand for USA military hardware.

Throughout all of this, it is important to keep in mind that the USA privilege of being the printer of the global currency is and remains the mechanistic backbone of the global empire, ever since the 1971 end of the Bretton Woods system. The US dollar retains its status via international demand for the US dollar, which, in turn, comes from USA control of the main commodities that have the greatest global demand and the highest prices in US dollars. These dollar-boosting “commodities” include: oil and gas, opium, financial debts of nations (serviced in US dollars), US-dollar currency choice to secure savings and investments, and USA military hardware. Recently, the USA is proposing exorbitant rent extraction (in US dollars) for its globally distributed military bases.[71]

Oil and gas are tricky, because Russia, Venezuela, China… have oil, gas, coal… and because the USA domestic energy sector (shale) is developing, causing a glut, lower energy prices, and less demand for the US dollar. Whereas a high price of oil helps USA shale, it also helps global opponents Venezuela, Iran and Russia. One “solution” is military or financial (“sanctions”-based) destruction of all energy-producing centers that the USA does not control, which may be present USA strategy?

In all of this, the Western middle and professional classes must consent (by agreement or inaction), be wilfully blind to what is actually going on, and keep “hope” in their politicians and the future. The next sections describe the vast social engineering campaigns that were created following the fall of the Soviet Union. It is not generally appreciated that these campaigns were

massively organized and implemented immediately following the fall of the Soviet Union. The said campaigns installed a primacy of select social concerns, thus masking the actual cultural and social-class restructuring, for those influential classes that can afford the illusion. Selected, siloed and constructed social concerns were: gender equity, anti-racism, and global environmentalism.

PART-II: SOCIAL-CONSTRUCT GLOBALIZATION

Mass-cooperation induced by organized religion

« [P]our qui est seul, sans dieu et sans maître, le poids des jours est terrible. Il faut donc se choisir un maître, Dieu n'étant plus à la mode. »[4] —Albert Camus, *La Chute*, 1956

A successful empire-sanctioned and supported religion is a powerful vehicle to direct individual impetus and self-image, thereby stabilizing the empire against both rebellion and worker lethargy.

Nationalism itself is such a religion, but it can fall out of favour in an ethos of international globalization for the greater good. The Roman Empire had Roman Catholicism, which later infused European colonial powers. "The Gods" that surveil citizens for moral rectitude have often found their homes in states and empires.[72]

Such policy considerations were saliently brought forth in the early 1970s, as the instabilities from the post-Bretton-Woods globalization were first becoming palpable. The Trilateral Commission think tank was founded by David Rockefeller in 1973, and its most influential report is "The Crisis of Democracy", published in 1975.[7] The report is silent on the Bretton Woods dissolution catastrophe and the emergent globalization, yet it expresses newfound concern for managing democratic societies. The report's authors explain the need for "gods" this way (at pages 159-160):

> "—What is in short supply in democratic societies today is thus not consensus on the rules of the game but a sense of purpose as to what one should achieve by playing the game. In the past, people have found their purposes in religion, in nationalism, and in ideology. But neither church, nor state, nor class now commands people's loyalties. […] But now all three gods have failed. We have witnessed the dissipation of religion, the withering away of nationalism, the decline— if not the end— of class-based ideology.
>
> In a nondemocratic political system, the top leadership can select a single purpose or closely related set of goals and, in some measure, induce or coerce political and social forces to shape their behavior in terms of the priorities dictated by these goals. […] In a democracy, however, purpose cannot be imposed from on high by fiat; nor does it spring to life from the verbiage of party platforms, state of the union messages, or speeches from the throne. It must, instead, be the product of the collective perception by the significant groups in society of a major challenge to their well-being and the perception by them that this challenge threatens them all about equally. […] Now, however, these purposes have lost their salience and even come under challenge; the imperatives of

[4] Translation from the French: "[F]or anyone who is alone, without god and without a master, the weight of days is dreadful. Hence one must choose a master, God being out of style."

> national security are no longer obvious, the desirability of economic growth is no longer unquestioned.—”

The question arises: what can serve as an overarching religion (or collection of religions) that will support and stabilize increased USA global economic predation in the unipolar context following the fall of the Soviet Union, in a globalized world built on “universal human rights” since the end of WWII, in which multicultural immigration is a labour-supply reality?

The devices and illusions of “human rights” and “democracy” worked well for decades but it is difficult to maintain these constructs in a world in which globalization is more aggressive, more extensive, and visibly more violently enforced. Furthermore, the Cold War is no longer much of a unifying threat for Western populations.

After being subjected to the September 11, 2001 attacks, initiating the war against Afghanistan, and opening Guantanamo Bay, on May 6, 2002, the USA withdrew its signature from the 1998 *Rome Statute of the International Criminal Court*, which established four core international crimes: genocide, crimes against humanity, war crimes, and the crime of aggression.

The empire seeks to turn our attention away from actual crimes with actual victims — whether the weapons are depleted uranium or economic sanctions or debt devastation or capital flight — and instead asks us to look up to the sky for the threat (CO2) that could end the human species, no less, unless we are sufficiently good, active, and cooperative.

This, in our opinion, is the process of how the global-warming “religion” was born. Like any proper religion of an empire, it must be taxable, exploitable by a large layered array of power players, and useful in motivating massive restructuring campaigns. The alleged danger must be gigantic, involving humanity and the planet itself, in order to focus attention, and for personal investment in the religion to be rewarding.

The following section presents data showing that the current global warming ethos was artificially created following the 1991 fall of the Soviet Union, and later exploited by global financiers in the mid-2000s to create carbon trading and a carbon economy.

If there is any doubt of the potential for the global warming paradigm to in-effect be a State “religion”, even justifying war, the words of Noam Chomsky, spoken in 1994 to 1996 and 1999, merit being noted:[73]

> “—For example, suppose it was discovered tomorrow that the greenhouse effect has been way underestimated, and that the catastrophic effects are actually going to set in 10 years from now, and not 100 years from now or something. Well, given the state of the popular movements we have today, we’d probably have a fascist takeover—with everybody agreeing to it, because that would be the only method for survival that anyone could think of. I’d even agree to it, because there just are no other alternatives around right now.—”

Following the global-warming section, further sections will present similar data regarding gender equity and anti-racism, as state ideologies. If the reader finds it difficult to consider that ideology related to gender and anti-racism can be a surrogate State religion, then we invite them to note how Russia has in recent years expressly motivated developing policy, ratifying national statutes, and lobbying the United Nations, to enshrine “family values” and gender-role

preservation, as questions of sovereignty, national security, and societal stability. It is interesting that, among others, Western gender-studies academics are pointing this out regarding Russia.[74]

Emergence, capture, promotion and institutionalization of global warming

We are not the first to propose that global warming is a religion. It seems the first was Alexander Cockburn, in 2007,[75] although he was preceded by Michael Crichton regarding environmentalism, in 2003.[76] A Google search for “global warming religion” presently gives over ten thousand relevant results. One recent example is this one.[77]

In a 2017 encyclopedia article, Mike Hulme writes:[78]

> “—The growing political resonance of climate change was partly explained by the dissolution of the Soviet Union between 1989 and 1991. Fears of Cold War destruction were displaced by those associated with climate change, prompting the observation at the time from cultural theorist Andrew Ross that, “apocalyptic fears about widespread droughts and melting ice caps have displaced the nuclear threat as the dominant feared meteorological disaster” (Ross 1991, 8).—”

This is correct, as can be seen in United Nations frameworks, and as the graphs below illustrate.

The international legal structure to monitor and control CO2 emissions was created immediately following the 1991 fall of the Soviet Union:[79]

> “—The United Nations Framework Convention on Climate Change (UNFCCC) is an international environmental treaty adopted on 9 May 1992 and opened for signature at the Earth Summit in Rio de Janeiro from 3 to 14 June 1992. It then entered into force on 21 March 1994, after a sufficient number of countries had ratified it. The UNFCCC objective is to "stabilize greenhouse gas concentrations in the atmosphere at a level that would prevent dangerous anthropogenic interference with the climate system".—”

The Earth Summit in Rio de Janeiro was an integral part of the United Nations (UN) (USA) response to the Soviet Union. The official UN “Introduction” text presenting a series of post-1991 world conferences, including the Earth Summit, has:[80]

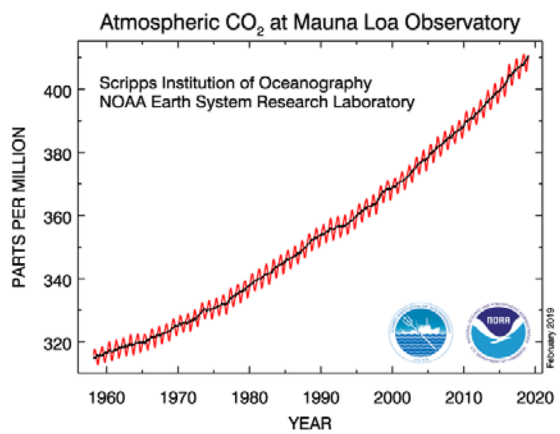
> “—All were convened with the strong support of the UN General Assembly, currently the voice of 185 Member States, and the recognition that the end of the cold war presented the opportunity — indeed, the necessity — to revitalize international cooperation on development issues. All addressed problems of a global magnitude which Member States recognized had grown beyond their individual capacities to solve and which needed a concerted international effort. All of them reflect the work of Member States and a growing number of other actors in the field of international development, particularly non-governmental organizations (NGOs). All of them actively sought out media attention, capturing the imaginations of millions of people around the world and greatly enhancing awareness and understanding of the issues in the public at large. —” (emphasis added)

At the same time, the pre-existing (1988) Intergovernmental Panel on Climate Change (IPCC) was assigned to assessing the science related to climate change specifically for use by the UNFCCC.

The formalized institutional backing at the highest levels, the involvement of sectors of civil society (NGOs), and the media coverage, instantly gave the global warming narrative a large boost, both in the amount of scientific activity and in the cultural and media realms (see graphs below).

The said boost was artificial, in that the planet did not suddenly experience an onslaught of sustained climate and weather catastrophes in December 1991. There was no global change of atmospheric or climatic regime in 1991. There was no sudden increase in atmospheric concentration of CO2 in 1991.

On the latter point, high-quality instrumental measurements of CO2 have been available since the 1950s:



Likewise, climatologists did not, in 1991, suddenly start using climate models to simulate the effects of increasing CO2, or suddenly develop more sophisticated global circulation models. On the contrary, radiative and heat-transfer atmospheric physics and global circulation models of the planet were essentially as advanced as they are today as early as the 1960s, and were being used to make essentially the same CO2-effect predictions as today.[5]

In 1967 leading theoretical climatologists Manabe and Wetherald calculated a 2 degree C increase in mean global near-surface temperature from a doubling of CO2 atmospheric concentration.[81] No one batted an eye. The media was silent.

Such calculations of surface-temperature sensitivity to CO2 and other factors quickly became a mature field of science, which was reviewed by Ramanathan and Coakley in 1978. Then, as now: “The principal weakness of the current models is their inability to simulate the feedback

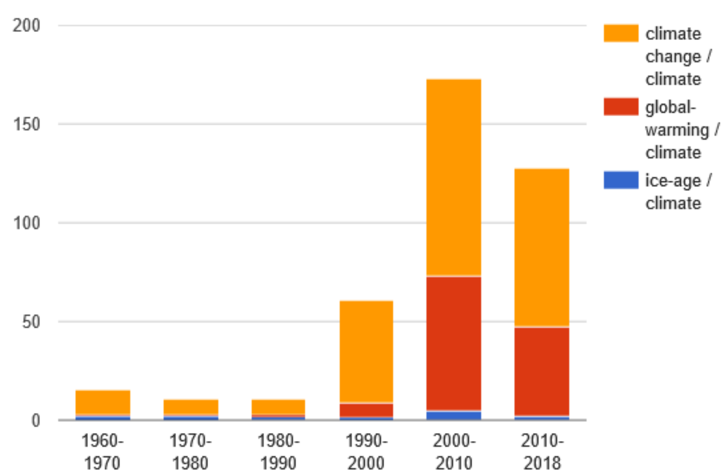
[5] And these model calculations suffered from the same shortcomings as they do today: unknown cloud response, unknown atmospheric water-vapour response, unknown “dirty snow” albedo response, unknown surface-humidity emissivity response, etc. See Rancourt for definitions of the basic concepts, in the radiation-balance context: “Radiation physics constraints on global warming: CO2 increase has little effect”, by Denis Rancourt, *archive.org*, 4 June 2011.
https://archive.org/details/RadiationPhysicsConstraintsOnGlobalWarmingCo2IncreaseHasLittleEffect

mechanism between surface temperature and cloud cover." The questions and the state of knowledge were essentially the same as today: "The review also summarizes radiative-convective model results for the sensitivity of surface temperature to perturbations in (1) the concentrations of the major and minor optically active trace constituents, (2) aerosols, and (3) cloud amount."[82]

Coupling of the ocean and atmosphere systems was included in global circulation models in 1969.[83] By 1980, detailed simulations of spatially-resolved earth surface warming were being produced. For example, Manabe and Stouffer reported winter warming in the range 6 to 18 degrees C for the Arctic Ocean and its surroundings, from quadrupling CO2 concentration (their figure 16).[84] No one got excited about a coming end of the world, whatsoever, not even when relative newcomers James Hansen and colleagues at the NASA Institute for Space Studies concluded in more alarmist terms in their 1981 paper in the influential journal *Science*:[85]

> "—The global warming projected for the next century is of almost unprecedented magnitude. On the basis of our model calculations, we estimate it to be ~ 2.5°C for a scenario with slow energy growth and a mixture of nonfossil and fossil fuels. This would exceed the temperature during the altithermal (6000 years ago) and the previous (Eemian) interglacial period 125,000 years ago (53), and would approach the warmth of the Mesozoic, the age of dinosaurs.—"

Likewise, climate and environmental scientists did not flock to research the coming CO2-induced possible end of the world. This flocking of direction in scientific research did not occur until the UN's post-Soviet-Union new-found UNFCCC concern to "stabilize greenhouse gas concentrations in the atmosphere at a level that would prevent dangerous anthropogenic interference with the climate system", and until the media hype surrounding the Earth Summit. The onset of the said flocking is seen in our Google Scholar search results:



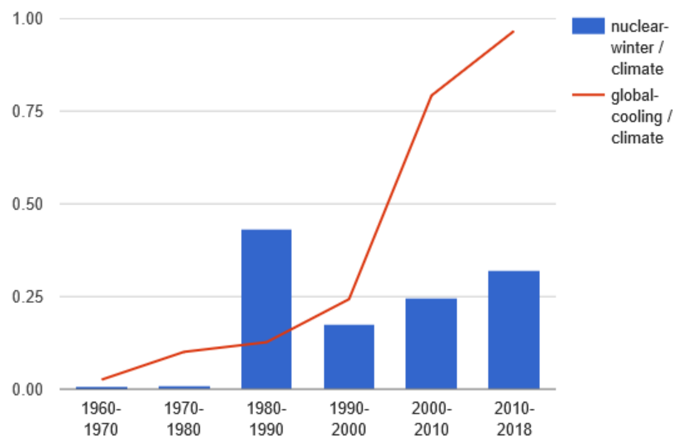
This chart shows the number of scholarly articles with the exact phrase "climate change" (orange), "global warming" (red), or "ice-age" (bleu), each divided by the number of articles with "climate", expressed in percentages, in decadal bands from 1960 to present. 100 % corresponds to approximately one million articles per decade.

In 2000-2010, 68 % of the scientific articles with “climate” had “global warming”; which is a surprisingly high number that would never occur from spontaneous organization of scientific truth-seeking without political influence, in our opinion.

Such a high degree of polarization of scientific research, we suggest, was produced by two mechanisms: alignment of scientific public funding-agency goals with the goals of the UNFCCC, and the media and societal-status appeal of the topic. Many of the scientists, in turn, and their professional associations, were also public-policy and media-commentator contributors, which is a positive feedback for the cultural acceptance of the topic.

A separate case study of science-society feedback amplification can be made of the “nuclear winter” scientific saga of the 1980s. In the nuclear-winter saga, apocalyptic-fear interest was generated in the popular culture, during the Cold War era, using the same global circulation models developed since the 1960s to simulate the climate consequences of a large nuclear war. In 1983, Turco *et al.* published the prediction of their “nuclear winter” in the influential scientific journal *Science*, which led to a decadal flurry of scientific work.[86]

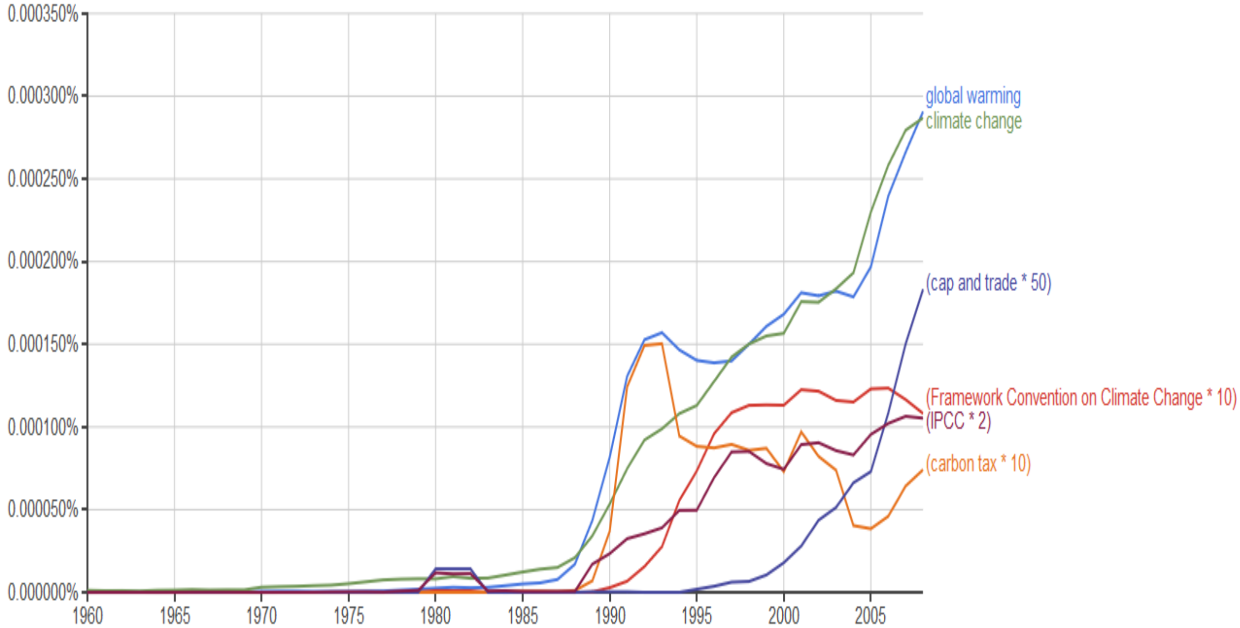
At the time, prediction of global cooling following a nuclear war seemed more worthy of increased attention than prediction of slow onset of permanent cooling from industrial activity. The difference with the continuing “global warming” episode is that “nuclear winter” did not have the backing of the UN or the USA finance and globalization interests. Nonetheless, the said feedback was qualitatively the same, and produced an instant onset of significant scientific activity, which died down at the onset of the “global warming” frenzy and never reached stratospheric proportions (pun unintended):



This chart shows the number of scholarly articles with the exact phrase “nuclear winter” (blue), or “global cooling” (red), each divided by the number of articles with “climate”, expressed in percentages, in decadal bands from 1960 to present. The topic “global cooling” is shown for comparison. 1 % corresponds to approximately ten thousand articles per decade.

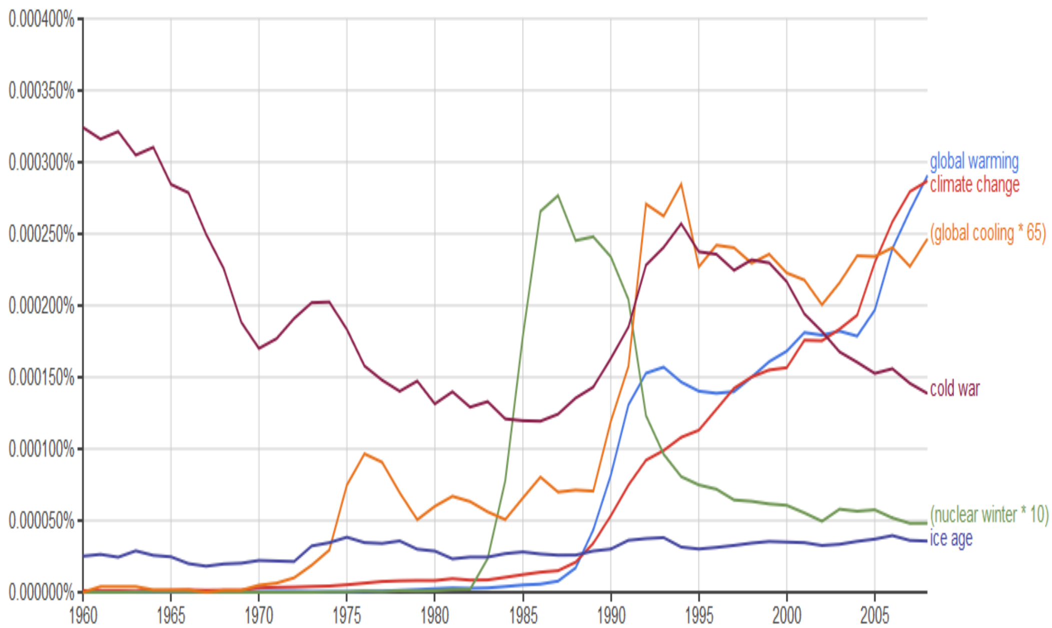
Sociologist Brian Martin has analyzed the degree of politicization of the science of nuclear winter predictions.[87] In 1990, Turco *et al.* reviewed the field of nuclear-winter predictions, and somewhat toned down their original prediction,[88] but there remained a “scientific consensus” that a large nuclear war would cause 10 degree C cooling.[89]

The rapid onset of scientific research into “global warming”, starting at the 1991 fall of the Soviet Union (above chart of Google-Scholar data), is also seen, at approximately the same year, as a broader societal phenomenon in the data of all published books, whether fiction or non-fiction; as is seen in our phrase-occurrence search of the Google Books “1960 to 2008, American English” corpus, using Ngram Viewer:



This graph shows the percentage of books (per year) in which the phrases “global warming”, “climate change”, “cap and trade”, “Framework Convention on Climate Change”, “IPCC”, or “carbon tax” occur. Some of the percentages are multiplied, as indicated, for ease in visualization. A smoothing range of one year was applied.

Next, we add the phrases “nuclear winter”, “cold war”, “global cooling”, and “ice age” for comparison (and remove some phrases for clarity):

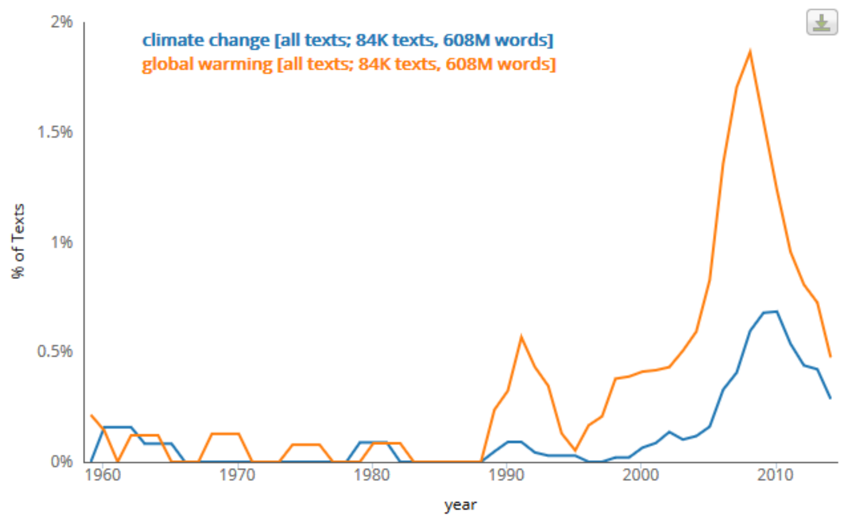


We see that “ice age” and “nuclear winter” were of more concern or interest than “global warming” and “climate change” up until the end of the 1980s. In the mid-2000s, “global warming” became of more concern or interest than “cold war”.

By 2008, “global warming” was of ten times more concern or interest than “nuclear winter” at its peak during the Cold War. That is phenomenal when one considers the imagery associated with a global nuclear war, the memory of Nagasaki and Hiroshima, the strength of the civil societal movements to reduce the risk of nuclear war, and the history of media attention for arms-reduction negotiations and protocols.

Societal concern or interest for “global warming” can also be measured by phrase-frequency data for the scripts (or generated captions) of all movies and TV shows. For this, we apply Bookworm, developed at the Cultural Observatory by Benjamin Schmidt and his collaborators.[6] Here is the result for “global warming” and “climate change”, 1960 to 2015:

[6] See the application here: http://movies.benschmidt.org/



This is for all 84 thousand texts of movies and TV shows. We applied a smoothing of one year. At its peak, “global warming” was present in a remarkable 2% of scripts of all movies and TV shows (on a per-year basis). For comparison, the word “homeless” has its maximum value of 5% after 2007. “Cold war” has a maximum value of 1.25% in the same year-range of 1960 to 2015 (in the vicinity of 2001). “Nuclear winter” never rises beyond 0.2% (in the vicinity of 1980).

In summary, all the reviewed data shows that “global warming” suddenly became “a thing”, both in the general culture and in the science community, when the UNFCCC and Earth Summit said it was a thing. Both the UNFCCC and Earth Summit were organized immediately following the fall of the Soviet Union.

This sudden “turning on” of “awareness” regarding an impending end of the human species from increasing atmospheric CO2 occurred at this late time even though virtually all the relevant science and its predictions (with the same limitations as today) had already been done and communicated by the end of the 1960s, by some of the same leading theoretical climatologists at the same theoretical climatology laboratories, such as Syukuro Manabe and Richard Wetherald. Other media-worthy catastrophe predictions from theoretical climatology, such as “nuclear winter” and emergence of the next ice-age, never attained the heights of “global warming” because they were not supported by the UN and USA globalization interests; even though the risk of nuclear engagement is objectively higher in periods of global instability such as during the 1990s fall of the Soviet Union.

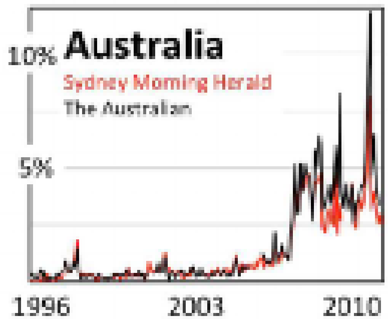
Carbon-trade rush of the mid-2000s

In addition to showing an early-1990s onset of preoccupation with “global warming”, the data from academic publishing (Google Scholar), all published books (Ngram), and movies and TV (Bookworm) also show a new large increase in “global warming” concern or interest in the mid-2000s (see graphs above).

The said mid-2000s increases are synchronous with the mainstream media becoming virtually monochromatic in its acceptance of global warming as a real and vital issue for humanity. Study of major daily newspapers across the world shows a large and discontinuous increase in media coverage of global warming or climate change occurring between the years 2005 and 2006. The

abrupt step-wise increase is typically four-fold in the magnitude of a newspaper's percent coverage for climate change (from 0.29 % in the years 2001-2005 to 1.26 % in the years 2006-2009, on average, on a per-year basis), and occurs simultaneously in all 27 countries studied, on all the continents: see Figure 1 and Table 3 of the article by Schmidt *et al.*[90]

This is the figure for the Australian data, from the Schmidt *et al.* paper:

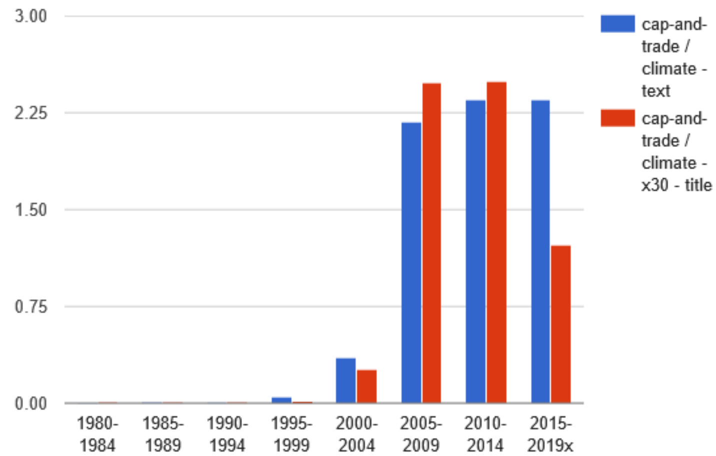


For these breaks in news-media coverage to be so large, so sudden and to occur simultaneously in all countries is not a phenomenon that is easy to explain, at least not in terms of the usual models of independent media outlets making newsworthiness decisions on the basis of authentic *a priori* readership interest.

There were no world climatic calamities in 2005-2006. Yet another IPCC report would not do it, as such reports never had. Former USA Vice-President (Democrat) Al Gore did put out his "An Inconvenient Truth" documentary film in 2006, but we see that as part of the media burst, not as a causal factor.

In looking for an actual cause of the media-coverage transition, one should have an eye to information about connections to global finance markets, such as the fact that "Generation Investment Management LLP (Generation IM) is a sustainable investment management firm, founded in 2004. It was co-founded by former US Vice President Al Gore and Goldman Sachs' Asset Management head David Blood."[91]

Regarding finance, the frequency of the phrase "cap and trade" shoots up after 2005 in our Ngram results from mining words in Google Books, shown in one of the graphs above. A sudden 2005-2006 increase in the frequency of the phrase "cap and trade" is also seen in our search of the Google Scholar database of academic articles, both in the texts of articles and in the titles of articles:



This chart shows the number of scholarly articles with the exact phrase “cap and trade”, in the texts of articles (blue), or in the titles of articles (red), each divided by the number of articles with “climate”, expressed in percentages, in half-decadal bands from 1980 to present. The “titles” data is multiplied by 30 for ease of visualization. The data for 2015-2019 was obtained by extrapolating the data for 2015-2018; that is, by multiplying the number for 2015-2018 by 1.25. 1 % corresponds to approximately five thousand articles per half-decade.

“Cap and trade” becomes a prominent topic in both all books and academic articles at the same time (2005-2006) that there is the dramatic and unprecedented increase in news media coverage for “climate change” or “global warming”, world-wide.

The late renowned historian of science and technology David F. Noble was acutely aware of the media explosion of the mid-2000s in Canada and the USA. In an important paper published on a blog in 2007, Noble wrote:[92]

> “— […] This potential for profit-making from climate change gained the avid attention of investment bankers, some of whom were central participants in the PCA through their connections with the boards of the Pew Center and Environmental Defense. Goldman Sachs became the leader of the pack; with its ownership of power plants through Cogentrix and clients like BP and Shell, the Wall Street firm was most attuned to the opportunities. In 2004 the company began to explore the “market-making” possibilities and the following year established its Center for Environmental Markets, with the announcement that “Goldman Sachs will aggressively seek market-making and investment opportunities in environmental markets;" The firm indicated that the Center would engage in research to develop public policy options for establishing markets around climate change, including the design and promotion of regulatory solutions for reducing greenhouse gas emissions. The firm also indicated that Goldman Sachs would “take the lead in identifying investment opportunities in renewable energy;” that year the investment banking firm acquired Horizon Wind Energy, invested in photovoltaics with Sun Edison, arranged financing for Northeast Biofuels, and purchased a stake in logen Corporation, which pioneered the conversion of straw, corn stalks, and switchgrass into ethanol. The company also dedicated itself “to act as a market maker in emissions trading” of CO_2 (and SO_2) as well as in such areas as “weather derivatives,” "renewable energy credits," and other “climate-related commodities.” “We believe," Goldman Sachs proclaimed, “that the management of risks and opportunities arising from climate change

> and its regulation will be particularly significant and will garner increasing attention from capital market participants."
>
> Among those capital market participants was former U.S. Vice President Al Gore. […]
>
> By the beginning of 2007 the corporate campaign had significantly scaled up its activity, with the creation of several new organizations. […] Also joining USCAP was the Natural Resources Defense Council, the World Resources Institute, and the investment banking firm Lehman Brothers whose managing director Theodore Roosevelt IV chaired the board of the Pew Center and was soon also to chair Lehman's new Global Center on Climate Change. As Newsweek now noted (March 12, 2007). "Wall Street is experiencing a climate change," with the recognition that "the way to get the green is to go green."—"

Therefore, we interpret the mid-2000s transition in "climate change" concern — seen to have occurred in a spectrum of academic and popular media and in national law-making initiatives (see [90]) — to have been caused by global financiers, based in the USA and connected to the Democratic Party. We postulate that these elite finance leaders have considerable sway, directly and indirectly, in the editorial policies of the major news media, especially the main trend-setting media in the USA and USA-allied or influenced economies. The scientists followed the funding and popularity trend.

In conclusion, whereas globalization following the demise of Bretton Woods and accelerated globalization following the fall of the Soviet Union were driven by USA hegemonic ambition itself, the said ambition seems recently to have aligned with beyond-Federal-Reserve USA investment banking opportunism, at least under the Democrats, with devastating consequences for local developing-world communities.[93] Furthermore, a new global "commodity" (carbon) traded in US dollars, under USA control of the global financial institutions, is one more commodity (with oil, opium, military hardware, and debt) to secure the US dollar as the world currency.

Emergence of gender-equity and anti-racism as state doctrines in the post-Soviet-Union era

Global warming is a powerful state-religion that has siloed concern and individual emotional investment away from the violence of globalization and class exploitation, including actual environmental destruction in the immediate environments of many communities, towards a diffuse danger for which everyone, and therefore no one, is responsible. It serves to appease the consciences of the professional-class collaborators, and of middle-class individuals who are vulnerable to privilege-guilt.

The first sign of a religious revolution against global warming as a dominant state ideology is the Gilets jaunes (Yellow Vests) movement that was sparked in France in 2018, which is fundamentally a class revolt (deplorables vs bobos-and-elites) that mirrors the Brexit vote and the Trump electoral phenomenon. Even former Canadian Prime Minister Stephen Harper, who oversaw the rise of the class conflict in Canada, has noticed and is now offering a non-solution of better managed conservatism, without addressing class-power-inequity, globalization or the USA problem.[94]

Two other state-religions are worthy of study, which arose following the fall of the Soviet Union, have attained extraordinary extremes, and which are experiencing backlash from the deplorables. The said two other state-religions are gender-equity and anti-racism, as siloed, limitless, and *de facto* class-blind doctrines.

The post-Soviet-Union rise of gender-equity and anti-racism as state doctrines followed a similar path as the rise of global warming: United Nations world-conference creation, academic-sector embrace, national statutory and institutional changes, broad media promotion, and cultural assimilation vectored by opinion leaders.

World Conference on Human Rights, 1993

In the case of gender-equity and anti-racism, the seminal UN world conference was the World Conference on Human Rights, held in Vienna, 14-25 June 1993. It was part of the flurry of UN-sponsored world conferences that were organized immediately after the fall of the Soviet Union, in "recognition that the end of the cold war presented the opportunity — indeed, the necessity — to revitalize international cooperation on development issues. [...] All of them actively sought out media attention, capturing the imaginations of millions of people around the world […]."[80]

The 1993 conference was the second-ever UN world conference on human rights. The first had been held in 1968. The main outcome of the conference was the "Vienna Declaration and Programme of Action" (VDPA), which was adopted by consensus by the 171 states present, including the USA. "As of today, all countries, except for Somalia and the United States of America, have ratified the Convention."[95] As recently as June 2017, a world general debate and progress report on the VDPA was hosted by the UN.[96]

The VDPA is a surprising document. On the one hand it rightly reaffirms the importance of universal human rights, and gives explicit assurances for the actual human rights of women, children, disabled persons, displaced persons, migrant workers, minorities, extremely poor persons, and victims of mass and war crimes, while on the other hand it represents significant departures from prior code regarding universal human rights.[97]

The said departures from prior code on human rights support globalization and support the implementation of frameworks leading to dubious state doctrines. We outline five such said departures prominent in the VDPA.

First, Chapter I, Section 31 of the VDPA reads:[97]

> "—31. The World Conference on Human Rights calls upon States to refrain from any unilateral measure not in accordance with international law and the Charter of the United Nations that creates obstacles to trade relations among States and impedes the full realization of the human rights set forth in the Universal Declaration of Human Rights and international human rights instruments, in particular the rights of everyone to a standard of living adequate for their health and well-being, including food and medical care, housing and the necessary social services. The World Conference on Human Rights affirms that food should not be used as a tool for political pressure.—"

This can be interpreted to condemn the use of unilateral so-called "sanctions" or trade blockades as political or military weapons but it does not use the words "sanction" or "blockade", nor is it clear that the purpose of the section is to prevent trade blockades by those states

powerful enough to apply such blockades. On the other hand, Section 31 has a distinct pro-“free trade” spin, where “any unilateral measure … that creates obstacles to trade relations among States”, such as national protective measures implemented by democratic states, is presumed to “[impede] the full realization of human rights … in particular the rights of everyone to a standard of living adequate for their health and well-being …”.

This is apparently the first time that a broadly accepted UN declaration and program of action links the attainment of basic “standard of living” to an absence of “any … obstacles to trade relations”, while not proposing any measures whatsoever to actually produce or allow development where development is needed. In this way, the VDPA anchors trade globalization in the attainment of alleviation of world poverty, at a fundamental conceptual level. To oppose “free trade” is to oppose human rights. The influence of the USA in the final text is palpable.

Second, Chapter I, Section 38 of the VDPA reads, in part:[97]

> “—38. The World Conference on Human Rights recognizes the important role of non-governmental organizations in the promotion of all human rights and in humanitarian activities at national, regional and international levels. […] Non-governmental organizations should be free to carry out their human rights activities, without interference, within the framework of national law and the Universal Declaration of Human Rights.—”

This was the first time that non-governmental organizations (NGOs) were assigned global reach and international rights in a broadly accepted UN declaration. This is remarkable because NGOs are non-government agencies, are not directly accountable to democratic state structures, and are easily influenced by large so-called philanthropists tied to powerful states.

Examples of said philanthropists, known to massively fund NGOs and associated think tanks, are the past and present “charity empires” of John D. Rockefeller, George Soros, Bill Gates …, which typically now purport to advance UN goals of health, democracy, and development (good governance, access to pharmaceuticals, access to technology). For example, there was no lack of funding for free economic advice to Russia after the fall of the Soviet Union.

Nowadays, few would deny that NGOs can be powerful vectors for interference, destabilization, undue legitimization, war propaganda and regime change, and that they are often tainted by geopolitical ambitions. This vector is statutorily supported by the VDPA, which instead, as a UN endeavor, should have guarded State sovereignty and international responsibility.

Third, Chapter II, Section 20 of the VDPA reads:[97]

> “—20. The World Conference on Human Rights urges all Governments to take immediate measures and to develop strong policies to prevent and combat <u>all forms</u> and manifestations of racism, <u>xenophobia</u> or related <u>intolerance</u>, where necessary by enactment of appropriate legislation, including <u>penal measures</u>, and by the establishment of national institutions to combat such phenomena.—” (emphasis added)

This is a stunning development. Nothing like this is present in prior major UN declarations or covenants. The text urges States to enact criminal statutory provisions against “phobia”, “intolerance”, and “all forms” of racism. The said urging is antithetical to longstanding international law that forbids criminalization of defamation, and that provides stringent conditions against state violations of individual opinion, belief and expression.[98]

Section 20 does not discriminate between racism of expression and racist actions against victims, nor does it discriminate between intolerance of attitude and actual denial of rights against victims. It encourages States to criminalize offending expression itself, thereby creating a chill against the very communication that is needed to resolve actual racial tensions in communities.

The VDPA's subchapter II.B.1 is entitled “Racism, racial discrimination, xenophobia and other forms of intolerance”, yet the subchapter and the entire VDPA are silent on the knowledge that racial tensions are spurred by economic pressures and aggression organized and manipulated by powerful players. Thus Section 20, in the context of the VDPA, displaces the problem from the causes to the symptoms, and seeks to irradiate the symptoms without addressing the causes, in a way that impedes people from potentially resolving conflicts by authentic expression of sentiment and emotion.

Section 20 is the seed for State penal enforcement of political correctness. It is an instrument for acceptance of “hate speech” laws, which are pathological laws that violently silence authentic individual expression, thereby pouring oil on any fire of racial tension.

Fourth, Chapter II, Section 39 of the VDPA reads, in part:[97]

> “—39. The World Conference on Human Rights urges the eradication of <u>all forms of discrimination</u> against women, <u>both hidden and overt</u>. The United Nations should encourage the goal of universal ratification by all States of the Convention on the <u>Elimination of All Forms of Discrimination against Women</u> by the year 2000. […]—” (emphasis added)

This is in the VDPA's subchapter II.B.3 entitled “The equal status and human rights of women”. The VDPA was the first time that the UN expressly enshrined women's right as a species of human rights. It put much emphasis on woman's rights, throughout.

The rights reviewed in the VDPA had some gender imbalance in their coverage. For example, the VDPA contains the phrase “girl-child” five times, but has no mention of the “boy-child” as soldiers, slave, or sex object. The document breaks from the practice of avoiding a gender-hierarchy of rights, and invites broad policy initiatives and State “education” as a corrective measure.

Finally, Chapter II, Sections 81 and 82 of the VDPA read, in part:[97]

> “—81. […] [T]he World Conference on Human Rights recommends that States develop specific programmes and strategies for ensuring the widest human rights education and the dissemination of public information, taking particular account of the human rights needs of women.
>
> 82. Governments, with the assistance of intergovernmental organizations, national institutions and non-governmental organizations, should promote an increased awareness of human rights and mutual tolerance. […] The proclamation of a United Nations decade for human rights education in order to promote, encourage and focus these educational activities should be considered. —”

This is in the VDPA's subchapter II.D entitled "Human rights education". Thus, the conference directs States to implement specific educational programs for human rights. In our view, since the States are the main purveyors and enablers of violations of human rights, this provision is disingenuous and was enacted for an ulterior motive. Western nations responded by creating more "social justice" education programs.

The new plans and intentions globally initiated in the VDPA have been vigorously and continuously pursued, with little result on the ground, one might add, and no method to quantify actual progress. One need only examine the most recent mass atrocities committed in the West's proxy war to destroy Syria, under the false pretext of attacking the very jihadist mercenaries that were funded and logistically and militarily supported by the West, Israel and their regional allies.[99][100][101] Another example is the ongoing genocidal attack in Yemen, using a hard blockade and continuous military bombing. Similarly, girl-child and women's basic human rights are difficult to find in Saudi Arabia or on Canadian indigenous reserves that have toxic water, almost three decades after VDPA.

Nonetheless, reality has not deterred purported good intentions. In a recent example, UN Women was created in 2010 and "is the United Nations entity dedicated to gender equality and the empowerment of women."[102] In its own words, listing its "major partners":[103]

> "—UN Women is thankful to all our business and philanthropic partners for their support of gender equality and women's empowerment.
>
> Some of our partners are Bill & Melinda Gates Foundation, Elizabeth Arden, Ford Foundation, Open Society Foundation, Procter & Gamble, PROYA Cosmetics Co. Ltd, Rockefeller Foundation, The Coca-Cola Company, Unilever, and Zonta International Foundation.—"

There is no lack of interest for gender equity, from the elite supporters of both globalization and trade in US dollars. What more proof does one need that the lives of women are improving thanks to the good will of USA global investors and merchants? There is also no lack of service collaborators to dress the illusion that such devices as UN Women advance human rights in the world.

Words that wound

The academic sector in the West embraced the new directions of the VDPA. Concomitantly with the World Conference on Human Rights, law academics wrote and published the highly (overly) influential 1993 book entitled: "*Words that Wound: Critical Race Theory, Assaultive Speech, and the First Amendment*".[104] This spawned the large new area of academic study known as "critical race theory", or at least was its legal-argument front.

Words that Wound was incisively criticized by Henry Louis Gates Jr.,[105] but this did not slow the enthusiasm for its radical new proposals that demolished the adage "sticks and stone may break my bones but names will never hurt me". The authors of *Wounds that Wound* have largely won, as more and more statutes and codes of behaviour enforce political correctness, and as many Western nations consider "denial" of State-approved versions of history to be penal offences that are systematically prosecuted.

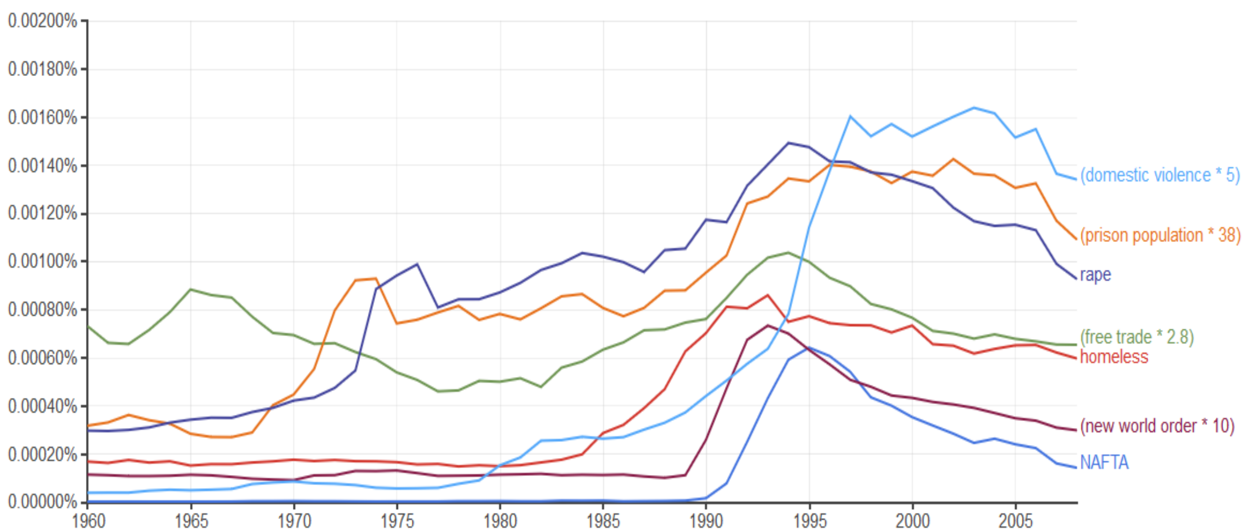
It's not just the questioning of the number of Jews who were murdered during the Nazi holocaust. Several influential pundits have in all seriousness called for jailing "climate deniers", as criminals against humanity.[106][107] To argue against State doctrine is to commit the new crime of "hate speech". A "hate crime" use to be a crime motivated by hate, as might be ascertained by the charged person's words. Now, the words themselves are the crime that routinely puts into motion the state's penal apparatus.[98][108]

University gender studies and critical race theory

The early 1990s saw the emergence of Third Wave Feminism, in which academic (institutionalized) feminism repositioned itself in the new "critical race theory" context of the post-Soviet-Union world. College departments of "women's studies" largely became departments of "gender studies",[109] and everyone became aware of "intersectionality" ("the complex, cumulative way in which the effects of multiple forms of discrimination - such as racism, sexism, and classism - combine, overlap, or intersect especially in the experiences of marginalized individuals or groups", *Merriam-Webster*).

More and more study programs became imbued with "critical race theory", and there were more and more social justice and gender studies programs. The social sciences and history and geography and English departments could not get away with being "old fashioned". Everyone needed to check their privilege. The burden of students (deemed oppressed) to fight their own oppression became an unjust burden that the State needs to assume.[110]

Book phrase-mining statistics show these trends in the academic culture, just as they illustrate author-perception of the new post-Soviet-Union globalization. First, the following graph shows indicators of globalization and related societal concerns.



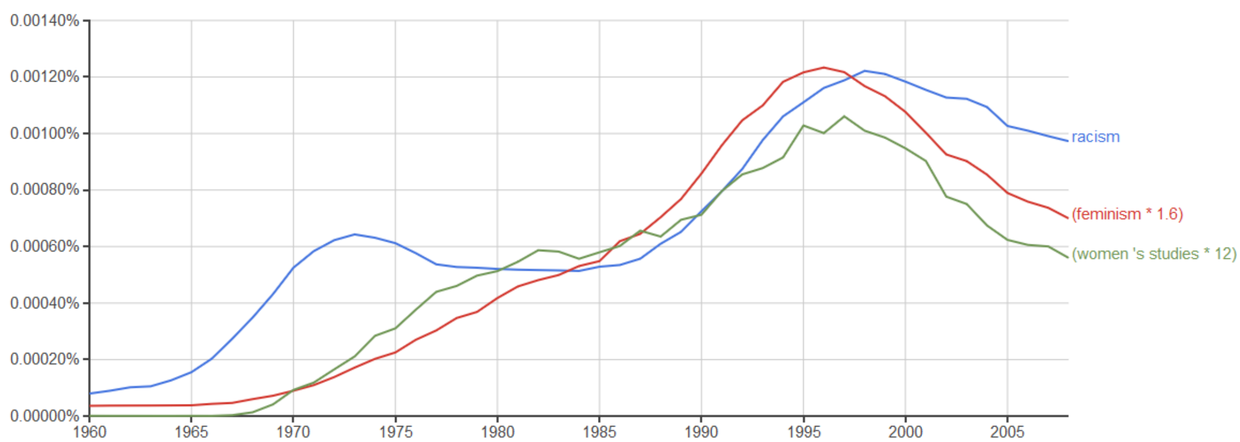
This graph shows phrase-occurrence frequencies for the Google Books "1960 to 2008, American English" corpus, using Ngram Viewer. The graph shows the percentage of books (per year) in which the phrases "domestic violence", "prison population", "rape", "free trade", "homeless", "new world order", and "NAFTA" occur. Some of the percentages are multiplied, as indicated, for ease in visualization. A smoothing range of one year was applied.

Here we see that “new world order” and “NAFTA” show sharp changes chronologically related to the 1991 fall of the Soviet Union. The phrase “free trade” has a large increase in slope at 1990, and its maximum in frequency of use occurs just following the fall of the Soviet Union, but it is otherwise a generally prevalent term throughout the entire period 1960 to 2008. The phrase “homeless” starts its rise from a steady background value in the 1980s, when the real phenomenon of urban homelessness became prevalent in the Western world (see above).[9] The phrase “domestic violence” starts to have a significant frequency in the early 1980s, has a large positive slope from 1986 to 1992, and has a sharp rise to a high-plateau value, which is synchronous with the increase of “NAFTA”.

The terms “prison population” and “rape” appear as signatures of aggressive globalization. They both have a step-wise increase following the USA’s 1971 unilateral cancellation of the Bretton Woods system, and they both have and additional step-wise increase following the 1991 fall of the Soviet Union. The same occurs with the phrase “sexual assault” (not shown). This suggests the idea that societal concern or attention for sexual crimes and criminality increases with increasing negative effects from predatory investment globalization (see Part-I, above, for said negative effects).

Similarly, phrase-frequencies of some of the broad topics relevant to the VDPA can be examined for changes in time. These phrase-frequencies show gradual and extended variations whereas other topics show sharp transitions chronologically associated with VDPA and the fall of the Soviet Union, as follows.

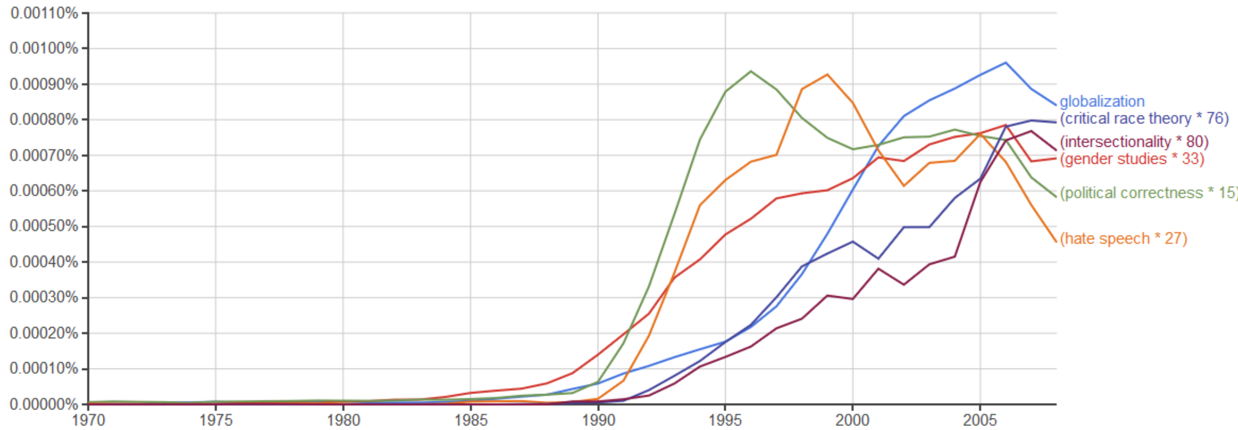
For example, this graph shows the percentage of books (per year) in which the phrases “racism”, “feminism”, and “women’s studies” occur:



Two of the percentages are multiplied, as indicated, for ease in visualization. A smoothing range of three years was applied.

Here, “feminism” and “women’s studies” increase after 1970, and again in the 1980s, but no change in behaviour or signature can be assigned to the 1991 changes in geopolitics.

However, new topics more specifically related to or spawned on the global scale in-part by the VDPA show a sharp and systematic post-Soviet-Union rise in their phrase-frequencies:

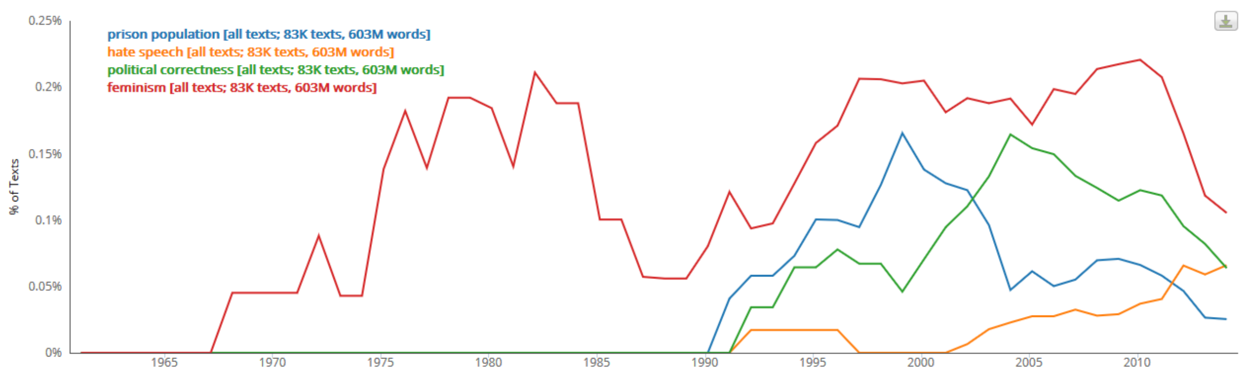


This graph shows phrase-occurrence frequencies for the Google Books "1960 to 2008, American English" corpus, using Ngram Viewer. The graph shows the percentage of books (per year) in which the phrases "globalization", "critical race theory", "intersectionality", "gender studies", "political correctness", and "hate speech" occur. Some of the percentages are multiplied, as indicated, for ease in visualization. A smoothing range of one year was applied. The range in years is 1970 to 2008, since the frequencies are virtually zero in the range 1960 to 1970.

Here, the near-1990 onset for all these phrases is striking, and coincides with the onset of the phrase "globalization". Basically, these terms or topics (critical race theory, intersectionality, gender studies, political correctness, and hate speech) became "a thing" in published books, starting at the 1991 fall of the Soviet Union, and the USA-led West's global response to the said fall.

The post-Soviet-Union onsets of societal concern or cultural emergences seen in published books are also detected in the phrase-frequency data for the scripts (or generated captions) of all movies and TV shows. For this, we again apply Bookworm (see above).

For example, "feminism" has a first rise following the 1971 collapse of Bretton Woods, and a second rise following the 1991 fall of the Soviet Union, whereas "prison population", "hate speech" and "political correctness" first emerge immediately at or following 1991:



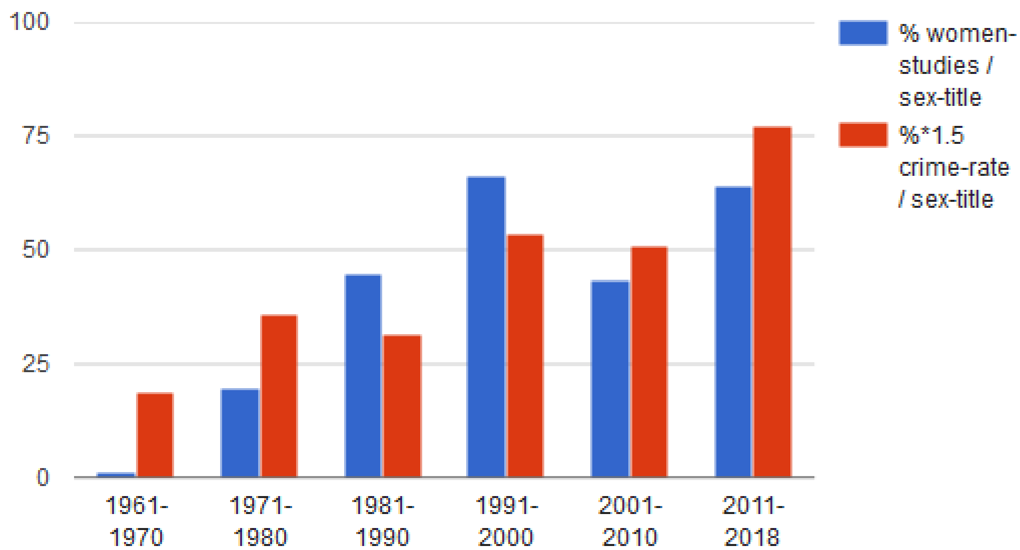
Here, a smoothing of 2 years was applied, for clarity in visualization. The y-axis is percent of texts or scripts in the entire data set, on a per-year basis.

Clearly, in the movies and TV, “political correctness” (like “hate speech”) became “a thing” immediately following the fall of the Soviet Union and the organization of the VDPA, accompanied by a new rise of “feminism”. Interestingly, “mental health” has a dramatic rise at approximately 1991, to a plateau of almost 1 % of all texts or scripts (not shown).

Analogous results arise in the academic-journal literature, as follows.

First, we examine the phrase-occurrence frequencies in the Google Scholar database, by decade, for the common phrases “women’s studies” and “crime rate”. We report the number of articles with the target phrase anywhere in the text, as a percentage of the number of articles in the same decade with the word “sex” in the title of the article.

The use of articles with the word “sex” in the title is simply a convenient normalization, which assumes that the fraction of articles with “sex” in the title is a constant fraction of the total number of academic articles in the decade, throughout the full time period in the graph. We multiplied the percentages for “crime rate” by 1.5, in order to facilitate visualization. 50 % on the y-axis corresponds to approximately 20,000 articles per decade:

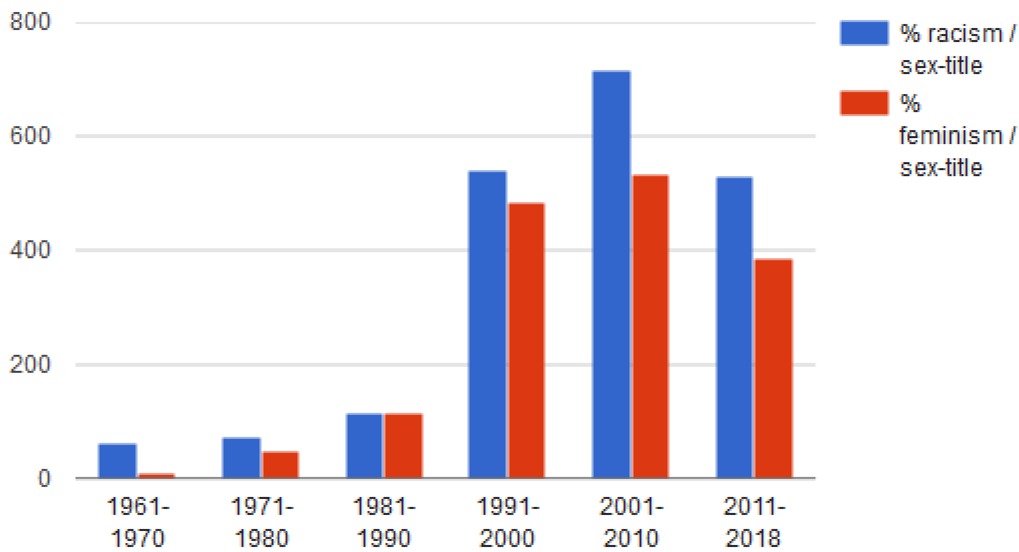


We note two features in the history of these target phrases. First, “women’s studies” starts in the 1970s, following the USA cancellation of the Bretton Woods system, when women’s studies programs were installed in universities, and experiences its next largest fractional increase (doubling) in the 1980s.

Second, “crime rate” phrase-frequency in academic journals — like “feminism” frequency in movie and TV scripts (above), and “prison population” and “rape” frequencies in published books (above) — is a signature of aggressive globalization. “Crime rate” has a step-wise increase following the USA’s 1971 unilateral cancellation of the Bretton Woods system, and an additional step-wise increase following the 1991 fall of the Soviet Union.

We interpret these “signatures of aggressive globalization” as follows. The globalization causes heightened levels of domestic economic pressure and social tension, thus increasing authors’ attention and concern for criminality, and sexual crime in particular, and attention and concern (in movies and TV) for the “feminism” response.

Second, we examine the phrase-occurrence frequencies in the Google Scholar database, by decade, for the common phrases “racism” and “feminism”. The same normalization, definitions, and method are used as above. 500 % on the y-axis corresponds to approximately 200,000 academic articles per decade:



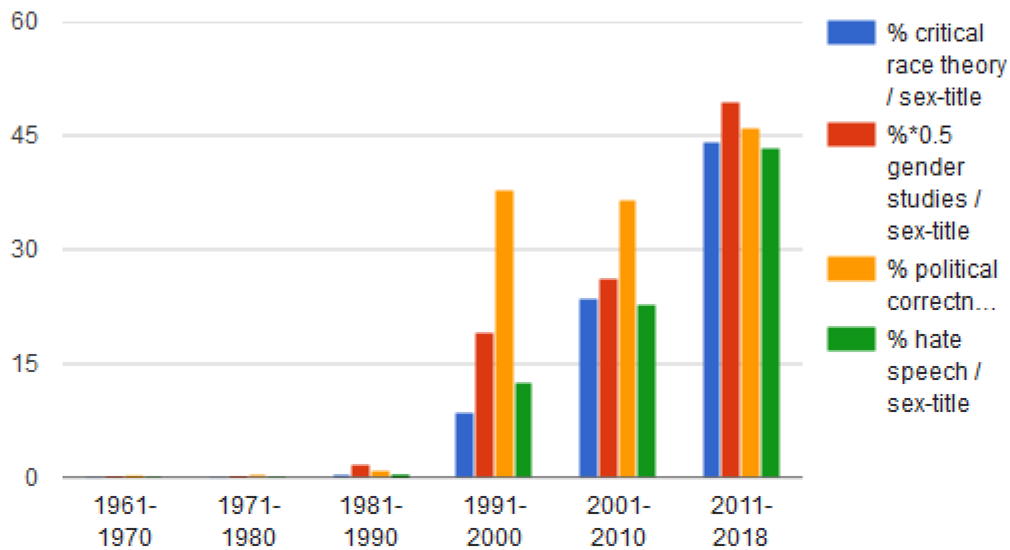
Like with “feminism” frequency in movie and TV scripts, “feminism” frequency in academic (Google Scholar) articles appears as a signature of aggressive globalization, turning on in step-wise fashion first at 1971-1980 (Bretton Woods cancellation), then at 1991-2000 (Soviet Union dissolution). Both steps correspond to approximately five-fold increases.

“Racism” occurs frequently throughout the Google Scholar database, but more so than with the books or movies and TV databases, “racism” in academic articles shows a sudden and sustained increase in the post-Soviet-Union and post-VDPA era. We see this as evidence that academics, as a service class, took their cue from and coalesced around the VDPA proposals. Actual racism regarding egregious violations of human rights did not, in the West, suddenly become a more urgent problem in 1991-2000.

By comparison, the massive post-9/11 (post-2001 World Trade Center attack) USA-led regime-change economic sanctions and civilian-infrastructure bombing war campaigns (Iraq, Afghanistan, Libya, Syria, Yemen) did not produce a comparable increase in “racism” concern among academics, nor did the extensive creation and use of drone bases under Obama. The post-9/11 war campaigns also did not lead to a flurry of UN world conferences, as did the 1991 fall of the Soviet Union. Not even a single UN wold conference has been convened to address present-era war campaigns. Instead, the UN is working feverishly to globalize and “share the burden” of refugee migration.

One does not have to be cynical to see the UN as the USA’s Uncle Tom, regarding propagandist ideological policy issues. The reality is hidden in plain sight, in virtually everything that the UN does; notably in the Earth Summit and the VDPA and all their offspring.

Third, we examine the phrase-occurrence frequencies in the Google Scholar database, by decade, for the phrases “critical race theory”, “gender studies”, “political correctness”, and “hate speech”, which we associate with the VDPA proposals. The same normalization, definitions, and method are used as above. The percent for “gender studies” is multiplied by 0.5 for improved comparative visualization. 50 % on the y-axis corresponds to approximately 20,000 academic articles per decade:



Clearly, all four topics enter the academic-journal corpus in 1991-2000. Three of the topics increase steadily with a large rate of growth (15 % of the “sex in title” reference per decade), whereas “political correctness” turns on abruptly and stays at a high level (approximately 40 % of the “sex in title” reference).

The four target phrases are not themselves present in the text of the VDPA, but they are the academic embodiment of the new direction expressed in VDPA. We see the four topics as related to the principle of freedom of expression, which itself is an internationally recognized human right.

The societal context of the developing gender studies[109] is one where it becomes disallowed by the State for an individual to “misgender” another individual, and where it can become statutory “family violence” for a parent to make “any attempt to persuade” their own child not to undergo gender-change medical treatment.[111]

For example, the Ontario Human Rights Code, in Canada, has:[112]

> “—The law recognizes that everyone has the right to self-identify their gender and that “misgendering” is a form of discrimination. […] Refusing to refer to a trans person by their chosen name and a personal pronoun that matches their gender identity, or purposely misgendering, will likely be discrimination when it takes place in a social area covered by the Code, including employment, housing and services like education. The law is otherwise unsettled as to whether someone can insist on any one gender-neutral pronoun in particular.—”

We view the measurable degradation of the right of freedom of expression, in general, beyond the confines of the said four topics, in the USA as a strong indicator of the degradation of democracy and social fairness in the USA, arising from the effects of globalization.[113]

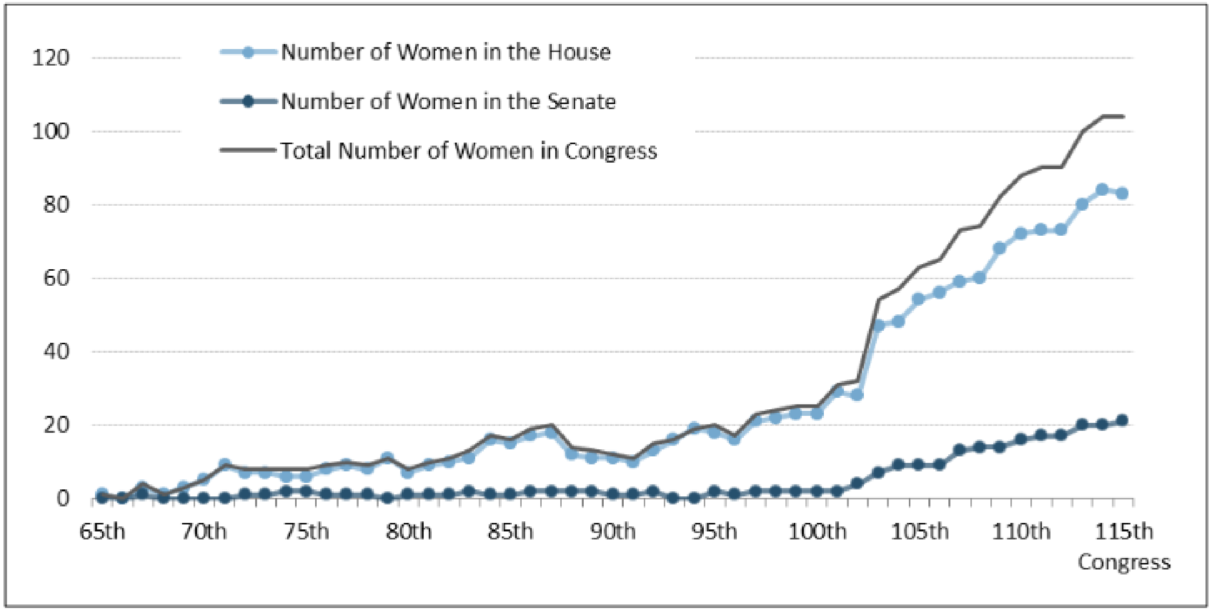
Women in Congress and Parliament

But propaganda and the maintenance of a mental environment and doctrine are not solely about media and academics. There is also a need to institutionally support the illusion of democracy. If the new equity doctrine can be meshed with an improved appearance of democracy, then the two fabrications support each other.

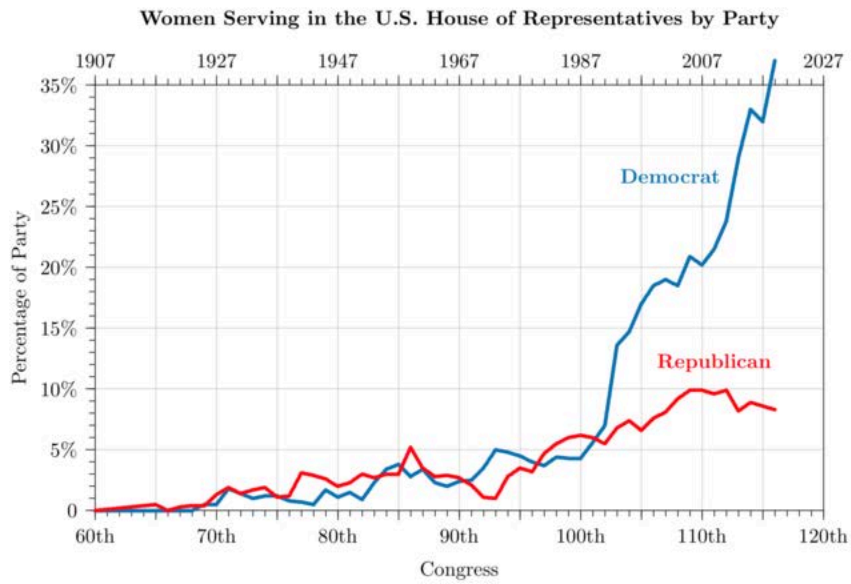
“The Year of the Woman was a popular label attached to 1992 after the election of a number of female Senators in the United States.”[114]

In the 103rd Congress of the USA, between 3 January 1993 and 3 January 1995, the maximum number of women members of Congress jumped from the prior Congress value of 32 to the then unprecedented value of 54.[115] The sharpness of the increase is seen graphically:

Figure 1. Number of Women by Congress: 1917-2017
Data for the 115th Congress are for the beginning of the Congress



The suddenness of the 1993 increase is seen most dramatically in the division between Democrat and Republican numbers for the House of Representatives:



Whereas the Republican and Democrat percentages followed each other and were both conservative numbers up until 1993, the Democrat numbers abruptly rose starting at 1993, the year of the UN VDPA promotion of gender equity in all spheres as a global ideal.

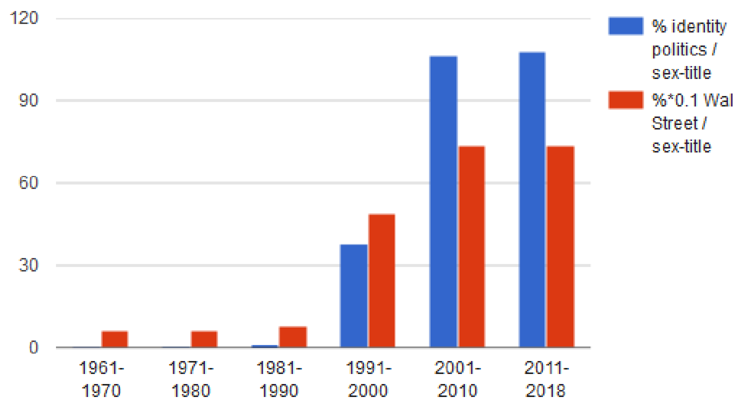
We interpret the difference in percentages of female House representatives between the two parties as arising in part because the Democrats are tied to global finance interests, which use the UN to shape acceptance of their practices, whereas the Republicans are tied to USA-based big industry (energy, military). Domestic energy has again become huge in the USA.[116] This division was spectacularly illustrated, on the USA domestic scene, in the 2016 election of Trump. In the words of Michael Hudson:[117]

> "—A new term was introduced to the English language: Identity Politics. Its aim is for voters to think of themselves as separatist minorities – women, LGBTQ, Blacks and Hispanics. The Democrats thought they could beat Trump by organizing Women for Wall Street (and a New Cold War), LGBTQ for Wall Street (and a New Cold War), and Blacks and Hispanics for Wall Street (and a New Cold War). Each identity cohort was headed by a billionaire or hedge fund donor.—"

These days, there is no lack of absurdity in having taken identity politics and the divisive equity maneuver ("equity" for a specific gender or race) to its logical end point: "Congress just Lynched another Black Woman in the Name of Anti-Racism."

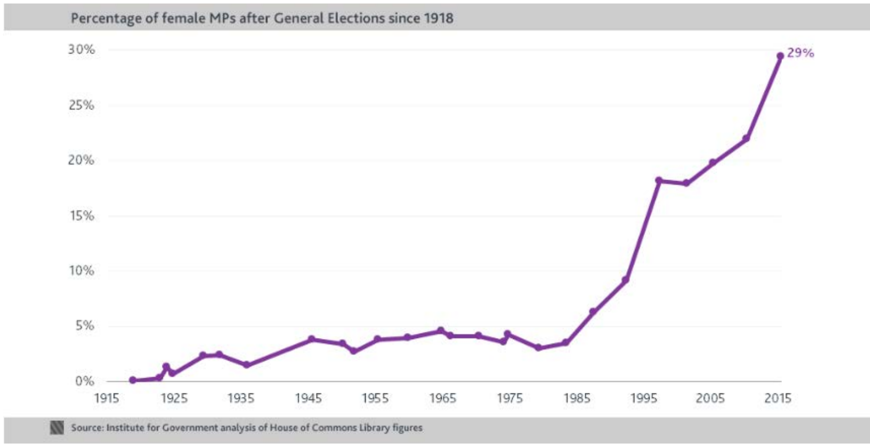


In short, prior to the present and recent Trump *et al.* backlash, the VDPA organized and heralded the birth of identity politics, newly minted to suit globalization in the post-Soviet-Union era. Such an important emergent phenomenon as “identity politics” needs to be bound by academics. Here is the phrase-frequency graph from Google Scholar, for the phrases “identity politics” and “Wall Street”, using the same method as described above:



“Wall Street” became of increased interest to academics in the new post-Soviet-Union era of globalization, starting in the 1990s, like never previously. Wall Street (private USA investment interests) was more involved in the new globalist expansion, compared to the post-Bretton-Wood era that was more purely about currency predation and classic national-debt extortion. “Wall Street” meshes with “identity politics”. The former is the new scammers. The latter is the new cover.

Increased woman as democratic figureheads, rather than solely men as democratic figureheads, was not limited to the USA. Here is a graph showing the large increase in percentage of female Members of Parliament in the UK, which occurred at 1993.



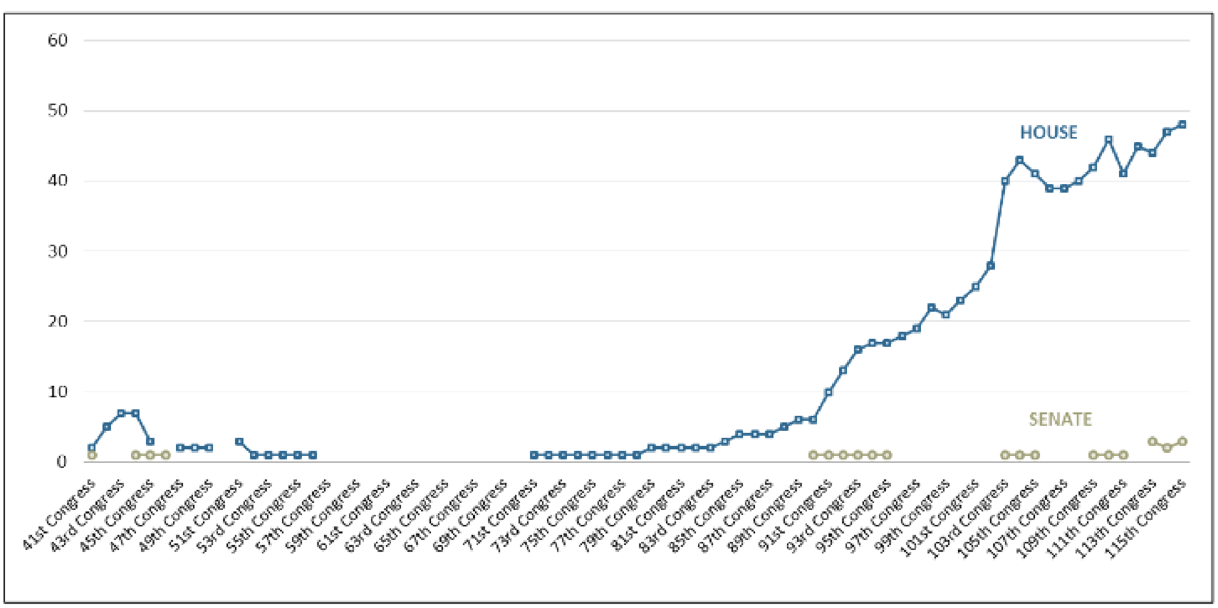
It would seem that voters in both the USA and the UK, an ocean apart, suddenly and spontaneously decided to elect female representatives, in synchronicity, in 1993?

African-Americans in Congress

The number of African-Americans in the USA House of Representatives appears to be a signature or indicator of aggressive globalization. It has a first rise immediately following the cancellation of Bretton woods (1971) to a plateau value of approximately 20, followed by a second rise immediately following the fall of the Soviet Union (1991) to a new plateau value of approximately 45:[118]

Figure 1. Number of African Americans in Each Congress, 1870 to Present

Including any Members who served only a portion of the Congress



We interpret this to mean that at times when the USA empire is emboldened to make a new thrust of predatory globalization and is confident that it is not threatened on the geopolitical stage in doing so, then it has shored up its number of African-American representatives to

bolster the appearance of democracy and social fairness. Furthermore, the second rise is aligned with the proposals of the VDPA.

These geopolitical trends invite us to consider whether the most recent new composition of Congress, “reflecting America’s diversity”, and including Muslim members, and a Palestinian member,[119] is related to the increased dispossession of Palestinians in the Middle East, covering up the vicious on-going wars against Syria and Yemen, and the continued attack against Iran by economic blockade, while explicitly threatening major wars in Iran and Venezuela. There will need to be increased Asian-American members of Congress if the USA increases its military moves against China.

Recent examples of State ideological excesses

With State ideologies, ideological enthusiasts are rewarded with promotions, praise, and positive media attention, which can lead to palpable excesses. Here are a few recent examples, which largely speak for themselves.

The respected Palestinian human-rights advocacy and Jewish media group *Mondoweiss* recently published two articles in which the authors position Israel’s militarized occupation — a longstanding human-rights regional hotspot that is characterized by a large apartheid gap in economic prospects, extreme State violence, a 10-year life expectancy gap, and a water consumption of Palestinians in the West Bank at 70 % of the WHO minimum, while being five times more for Jewish settlers[120] — in the context of the “climate crisis”.[121][122]

On February 4, 2019, David Klein writes to introduce his article:[121]

> “—The urgency of the global climate crisis makes it imperative for any social justice movement to come to grips with, and confront it in some way. There can be no social justice, after all, on a dead planet.—”

On March 12, 2019, Zena Agha entitles their article “Palestinians will suffer the impacts of climate change more severely than Israelis due to the occupation”, and introduces it as:[122]

> “—Climate change is among the greatest threats currently facing human life. Its effects are global, wide-ranging, and unequally distributed. Despite Palestinians and Israelis inhabiting the same physical terrain, Palestinians under occupation will suffer the effects of climate change more severely.—”

One wonders what the other “greatest threats currently facing human life” might be, if we were to put them on a scale of quantum of lost person-years per 1000 deaths, say?

Some professional academics also have had their say regarding climate change and the Israeli occupation.[123]

Regarding anti-racism ideology, in a March 11, 2019, article in the prestigious *Proceedings of the National Academy of Sciences of the United States of America* (*PNAS*), the authors explain the “Significance” of their work about “pollution inequity” as:[124]

> “—Some may find it intuitive that, on average, black and Hispanic minorities bear a disproportionate burden from the air pollution caused mainly by non-Hispanic whites, but

> this effect has not previously been directly established, let alone quantified. Our "pollution inequity" metric is generalizable to other pollution types and provides a simple and intuitive way of expressing a disparity between the pollution that people cause and the pollution to which they are exposed. Our results are timely, given public debate on issues relating to race, equity, and the regulation of pollution.—"

This was dutifully reported as highly newsworthy in the influential *Washington Post*, with the headline: "Whites are mainly to blame for air pollution, but blacks and Hispanics bear the burden, says a new study".[125]

Regarding the UN's continued role in promoting global warming and legalizing censorship, the three opening keynote speeches of the month-long UN Human Rights Council's 40th session in Geneva, pronounced on February 25, 2019, are worthy of study. As reported by the UN itself and echoed in the international media, the salient features of the three keynote speeches were as follows.[126]

The President of the UN General Assembly, María Fernanda Espinosa, raised those concerns that will not be acted upon, including "wars", and:[126]

> —Ms. Espinosa expressed concern about the widening gap between the planet's haves and have-nots.
>
> "Perhaps one of the most sensitive challenges for the human rights agenda is inequality," she said. "The concentration of wealth has increased to such an extent that, in 2018, 26 individuals had more money than the 3,800 million [3.8 billion] poorest people on the planet."—

"Michelle Bachelet, High Commissioner for Human Rights, highlighted the dangers of ignoring climate change:"[126]

> — "How can any State's interests be advanced by policies that damage the well-being of all humans?" she said. "This is true of climate change; you may know the saying, 'If you think economic interests are more important than environment, try counting your money while holding your breath.'"
>
> Ms. Bachelet also hailed the young climate activists inspired by Swedish teenager Greta Thunberg.
>
> The 16-year-old who had grabbed the attention of the world's media, recently travelled to the Davos World Economic Forum (WEF) in Switzerland where she called for the world's decision makers to take swifter action to limit carbon dioxide emissions and reduce global temperature rise to 2°C above pre-industrial levels.
>
> "In recent weeks I have watched children marching for sound climate change policies and other measures," the High Commissioner said. "As a parent, a grandparent and quite simply as a human being, they inspire in me a fierce determination to continue our struggle to uphold their rights."—

The UN Secretary-General's segment announces a global plan of action against "hate speech", and reads as follows:[126]

—**Hate speech ‘spreads like wildfire’**

In addition to improving women’s rights, the UN Secretary-General expressed alarm about the “shrinking civil space in every region of the globe”; and a rise in harassment, attacks and inflammatory rhetoric.

“Hate speech is a menace to democratic values, social stability and peace,” Mr. Guterres said. “It spreads like wildfire through social media, the internet and conspiracy theories. It is abetted by public discourses that stigmatizes women, minorities, migrants and refugees and any so-called ‘other’. Indeed, hate is moving into the mainstream, in liberal democracies and authoritarian States alike.”

To tackle this, the UN chief announced the creation of a fast-track strategy to scale up the organization’s response to hate speech and present a global plan of action, headed by his Special Adviser for the Prevention of Genocide, Adama Dieng.

This kind of initiative was necessary in light of the political capital earned at the expense of migrants and refugees, who some leaders had blamed for a rise in crime and terrorism, the Secretary-General insisted.

“We must re-establish the integrity of the international refugee protection regime and continue to work for common values and international cooperation to reassert rights and help protect people from ruthless traffickers, smugglers and other predators,” he said.—

Thus, the censorship desires of the “liberal democracies” will soon have explicit support in new UN policy. “Cyber bullying” cannot be allowed to make election results “unpredictable”. It seems: “Goodness must prevail.”

CONCLUSION

Take-home points are as follows:

- The Bretton Woods period (1945 to 1971) had regulated trade balances, regulated currency exchange, and a US dollar limited by being tied to gold. It was designed to develop the USA-led capitalist-block nations, against the communist bloc. It produced social-class-shared development and exhilarating social, cultural, engineering, and scientific advances. It worked too well. Japan, Western Europe and participating nations developed too much. The USA ended the Bretton Woods agreement in 1971 and started the first modern era of predatory globalization, with a second wave following the 1991 dissolution of the Soviet Union.
- “Globalization” is a euphemism for Western USA-led economic predation of countries in the so-called developing world, of the global under-classes as resources themselves, and of the Western USA-allied nations to the extent tolerable. From the USA perspective, the world is its plantation.
- The main administrative instrument for sustained global USA economic pillaging is the monetary instrument of the unbounded and USA-controlled US dollar as global currency. The said monetary instrument is essentially a conveyor belt for the continuous transfer of actual wealth and resources from the world to the USA system.

- Arguably, the main global concern of the USA, in addition to the classic geopolitical land-mass-resource and trade-route considerations, is to enforce and ensure, in tandem, the US dollar as the global currency.
- Enforcing the US dollar's status as the global currency includes covert and overt regime-change coups and wars — against administrations vying for currency sovereignty (sovereignty) — and economic and trade blockades, whereas "ensuring" the US dollar's status involves controlling major "commodities" to be purchased in US dollars, thus securing demand for the US dollar.
- The US-dollar-ensuring "commodities" to be controlled include: energy, opioid drugs, national debts of debtor nations (excluding the USA), monetary savings of the world elite (legally or illegally acquired), and USA military hardware and military bases ("protection") imposed on allied nations at exorbitant prices; and extend into the always developing globalized markets of pharmaceuticals (vaccines, etc.), GMO patented crops, and proprietary high technology (5G, etc.).
- Basically, the modus operandi of the USA Empire has been: any localized world mineral or essential resource of global importance will be controlled, through whatever means (military occupation, destruction of capacity, blockade, puppet regime…).
- Globalization is progressive and has occurred in bursts that define globalization eras. The first era was the post-Bretton-Woods era (1971-1991), starting when the US dollar was decoupled from gold.
- End results of the post-Bretton-Woods era were: the systematic relative loss of middle-class economic status, and palpable social misery in the West, such as the emergence of urban homelessness in the 1980s, associated with a predictable major Western recession (1982 crash, from Third World debt defaults that were written down via Brady bonds[29]).
- The second globalization era started immediately after the 1991 fall of the Soviet Union. It was a period of extended and accelerated globalization. The close targets were traditional USA-allied markets: Canada and Mexico (NAFTA), and Europe (mega-mergers). Europe somewhat resisted by forming the European economic union. Investment returns went into the stratosphere, as did CEO salaries. The USA industrial working class was decimated. China was brought into the capitalist orbit. The "deplorables versus bobos-and-elites" divide was created, as a major socio-geographic consequence in the West.
- Measured human consequences synchronous with the post-1991 acceleration of globalization, mainly affecting the lower-income classes, in the West, include: loss of welfare safety net, increase of number of single-parent families, threefold increase in rate of confrontational litigation in the courts, between parents and between individuals and with the state ("crisis in access to justice"), increased low-income household basic-need incidence (housing, health, safety, work, finance), increased rates of both suicide and suicide attempt, increased rate of opioid overdose (preceding the opioid epidemic of the 2010s), and increased rates of chronic asthma emergencies, and asthma prevalence, in both children and adults.
- Increased leniency in food and drug regulation, and a dramatic increase in the global use of the herbicide glyphosate starting in 1993 in the USA, were concurrent with post-1991 upsurges of diseases and chronic ailments: death from intestinal infections; incidence of thyroid cancer; death from Parkinson's disease; prevalence of diabetes; autism in children of different age groups; and phobia, anxiety disorder, panic disorder.
- The mid-2000s saw Wall Street and the major USA Banks take a more leading role in globalization, one that is eclipsing the traditional global economic instruments that are the World Bank and International Monetary Fund. The USA's so-called subprime

mortgage crisis, the 2008 crash, the mega-bailouts… are symptoms. The monkeys are demanding and being allowed more run of the zoo, in which all of the play is in US dollars.

- The large acceleration and expansion of globalization occurring immediately after the 1991 fall of the Soviet Union is not generally recognized as having been a USA response to the said fall, but it should be recognized as such. There was a large acceleration of globalization, both structural and in terms of extension and volume, and there can be little doubt that it was a response to the newly apparent geopolitical and ideological fracture.
- At the same time, in express response to the end of the Cold War, the UN undertook an unprecedented flurry of highly mediatized world conferences. Most notably, the UN advanced new paradigms of global concern that can be categorized as “climate change”, “gender-equity”, and “anti-racism”; and put in place declarations and plans to institutionalize and legalize these new paradigms of global concern.
- The said new paradigms of global concern are siloed and sanitized concerns, in-effect devoid of social-class, development-disparity, exploitation-structure and nation-sovereignty practical dimensions. They became global and state “religions” to pacify, hypnotize, and align populations for continued globalization, including the first steps towards a global carbon economy (with carbon traded in US dollars).
- The government, scientific, academic, education, NGO, and media sectors embraced and promoted the new paradigms of global concern. All globally-controlled corporations greened and equified. There could never be enough climate change prevention, gender equity, or racial social justice; and all problems and risks were due to deficits in climate change prevention, gender equity, and racial social justice.
- A social-justice education industry developed, based on newly-minted “critical race theory”, which transformed old-fashioned political analysis of exploitative power relations into awareness of “intersectionality”, and old-fashioned political analysis of social coalition formation into recognition of white privilege and the unjust burden of being brown.
- The UN had explicitly called for criminalization (“penal measures”) of “all forms and manifestations of racism, xenophobia or related intolerance”, and this elite-instigated desire was made reality with codes of conduct, vast internet censorship, hate-speech prosecutions, exploding defamation litigation threats, and arrays of sanctions against unapproved political views.
- The only effective resistance against globalization in the West has become the recent electoral and demonstrative revolts related to the Brexit vote, the Trump electoral victory, and the Gilets jaunes movement, all newly understood as the class conflict between the deplorables and the bobos-and-elites, between the sedentary rural inhabitants (the “somewheres”) and the globalist urbanites (the “anywheres”).
- Thus, it is no accident that the deplorables express their particular multi-faceted array of complaints from needed economic revitalization of the rural nation, to rejection of carbon taxation, to repudiation of the gender-equity and anti-racism programs, including censorship and political correctness.

Denis Rancourt is a Researcher at the Ontario Civil Liberties Association since 2015. He is a former Full Professor of Physics at the University of Ottawa, Canada.

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